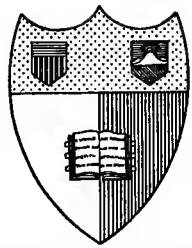
THE PSYCHOLOGY OF THE SPECIAL SENSES AND THEIR FUNCTIONAL DISORDERS

ARTHUR F. HURST

OXFORD MEDICAL PUBLICATIONS



THE CROONIAN LECTURES

ON

THE PSYCHOLOGY OF THE SPECIAL SENSES AND THEIR FUNCTIONAL DISORDERS

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"MIRACULOUS" CURE OF BLINDNESS AND HEMIPLEGIA

Reproduced from Carré de Montgeron's "La Vérité des Miracles," 1747

Frontispiece, see page 114]

THE CROONIAN LECTURES ON
THE PSYCHOLOGY
OF THE
SPECIAL SENSES
AND THEIR
FUNCTIONAL DISORDERS

DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS IN JUNE 1920

BY

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PREFACE

THE Croonian Lectures appeared in a shortened form in the *Lancet* soon after their delivery. They are now published in full with the addition of some illustrative cases.

I am anxious to acknowledge my great indebtedness to the Medical Officers who worked with me before and since the War at Guy's Hospital, and during the War in Lemnos, Salonica, Oxford, Netley and the Seale Hayne Military Hospital, as most of the investigations upon which these Lectures are based were carried out in conjunction with them, and the description of many of the actual cases are taken from their notes.

I wish especially to mention Major W. Johnson, M.C., Major J. L. M. Symns, Major J. F. Venables, Captain A. Wilson Gill, Captain R. G. Gordon, Captain G. MacGregor, Captain J. W. Moore of the U.S.A. Army Medical Service, Captain W. R. Reynell, Captain C. H. Ripman, Captain A. Robin, Captain S. H. Wilkinson, and Dr. R. Gainsborough.

The frontispiece has been reproduced from a copy of *La Vérité des Miracles* in the possession of the Royal Society of Medicine, by kind permission of the Librarian, Sir John Macalister.

ARTHUR F. HURST.

LONDON,
September 1920.

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CHAPTER I

NATURE OF HYSTERIA

When I received the honour of being invited to deliver the Croonian Lectures, it seemed to me right that I should attempt to apply some of the lessons I had learnt during the war to the problems of civil life. I was fortunate in having almost unique opportunities for investigating the neuroses, which occurred so frequently under the exceptionally trying conditions the British army had to face on every front. In the course of my investigations on their origin and nature I made a number of observations which throw, I believe, some light on the psychology of the special senses and the physiology of the reflexes associated with them. As the most common neurosis affecting the special senses was hysteria, and as my views on the nature of hysteria differ from those held by the majority of neurologists, it will be necessary to explain what I mean by hysteria before I approach the main subject of these lectures.

Nature and Definition of Hysteria.—The word hysteria was first used by the Greek physicians to describe what is known now as an hysterical attack, under the impression that it was due to an actual displacement of the uterus into various parts of the body. In course of time numerous other conditions were recognised as being of a similar nature, and the word hysterical was used to describe them all. The etymology of the word coloured all

the theories of the nature of hysteria up to the time of Charcot, almost every writer believing that it was associated with pelvic disorders and that it occurred only in women. Charcot, however, showed that it was not uncommon in men, and the earlier idea of its relation in women to pelvic disorders has been gradually discarded. The conception of the disease, which I shall use in these lectures, has nothing in common with the original one, and it might therefore be considered wise to use some other term than hysteria to describe it; but since this word has been accepted for so long a period by physicians in every country, it would be impossible to introduce any new term which would obtain universal approval. The word hysteria is indeed no more unsuitable than such words as rheumatism and chorea, which have also lost their original significance.

One common factor in all the symptoms, which are by universal consent regarded as hysterical, is their origin as a result of suggestion. When the history of each case is carefully investigated, it is almost invariably possible to discover that some event or condition suggested its onset and led to the exact form of the symptoms present. If the view be accepted that hysterical symptoms are caused by suggestion, they must be capable of removal by psychotherapy without assistance from any more material method of treatment. We can thus define an hysterical symptom as one which has been produced by suggestion and is curable by psychotherapy.

Physical Stigmata.—Gendrin, in an address to the Académie de Médicine in 1846, stated his belief that "hysteria is not only characterised by spasmodic attacks occurring at intervals, but it is a continuous disease, which in the intervals always presents characteristic symptoms," the most important of which "is a state of general or partial insensibility." Gendrin's views were to a great extent forgotten until they were revived by Charcot in 1871. Charcot taught that hysteria manifests itself in two ways—by persistent symptoms or stigmata, of which the patient is unaware, and temporary symptoms, which are obvious to the patient and lead him to seek medical advice. The former were supposed

to be present before the latter appeared and to persist after they had disappeared. Amongst these stigmata the most constant, according to Charcot, were retraction of the field of vision, certain forms of cutaneous anæsthesia, and pharyngeal anæsthesia. His teaching soon became universally adopted, and few books on neurology or general medicine published in the last fifty years fail to mention these stigmata.

One of Charcot's most distinguished assistants, Babinski, was the first to throw doubt upon the importance of the stigmata he described. Babinski [1] believes that they are not permanent symptoms at all, but, like the more obvious and temporary symptoms, are produced by suggestion. Instead of being produced by suggestion on the patient's part, they are the result of suggestion on the part of the physician. The evidence brought forward by Babinski in connection with anæsthesia and retraction of the field of vision appears to me to be conclusive, and receives further confirmation from the investigations which I shall presently describe. We have obtained similar evidence with regard to pharyngeal anæsthesia, so that the question of the physical stigmata may be regarded as settled.

Mental Stigmata.—It is so common to regard certain mental qualities as hysterical, and to apply the term hysterical to a certain type of individual, that it requires considerable courage to reject altogether the doctrine of a specific psychical disorder to which the name hysteria can be given. But on examining the literature, it is at once apparent that no sort of unanimity exists as to what are the specific mental attributes of hysteria. For several years I accepted the definition of hysteria as an abnormal mental condition, in which the individual is unduly prone to develop symptoms as a result of suggestion. But the experience of the war has taught us that, given a sufficiently powerful suggestion, there are probably no individuals who would not develop hysterical symptoms. [2]

Abnormal suggestibility, like other mental attributes commonly regarded as characteristic of hysteria, is undoubtedly a most important predisposing cause of hysterical symptoms, but it is not an essential factor, and cannot

therefore be regarded as an essential part of hysteria. As it may exist in an individual, who never shows any hysterical symptoms owing to the absence of adequate exciting causes, abnormal suggestibility cannot be regarded as a disease, such as hysteria would designate, any more than irritability or any other mental attribute is a disease. Unless everybody can be regarded as a victim of hysteria, as everybody is liable under sufficient provocation to develop hysterical symptoms, suggestibility is not the mental basis of hysteria. Whether a given person will develop hysterical symptoms under given conditions depends on the degree of his suggestibility and the strength of the suggestion. It is clear, therefore, that abnormal suggestibility is simply a predisposing factor, and is no more a part of hysteria than a tuberculous family history is a part of tuberculosis. Many cases of gross hysterical symptoms occurred in soldiers, who had no family or personal history of neuroses, and who were perfectly fit until the moment that one of the exceptionally powerful exciting causes, such as occur comparatively rarely apart from war, suggested some hysterical symptom. After its disappearance as a result of psychotherapy the man was once more perfectly fit, and his subsequent history showed that he remained no more liable than any of his companions to develop new symptoms.

As soon as it is recognised that the mental stigmata, which predispose to the development of hysteria, are not themselves a part of hysteria, it becomes obvious that many cases of hysteria will be missed if it is only looked for in so-called hysterical persons. When, on the other hand, it is remembered that there is nobody who may not develop hysteria if the provocation is sufficiently great, it must follow that hysteria is infinitely more widespread than has generally been supposed.

We thus arrive at the conclusion that, apart from actual hysterical symptoms, there are no underlying physical or mental symptoms or groups of symptoms, which precede and accompany them and persist after their disappearance, to which the term hysteria can be applied. It is clear, therefore, that while it is easy to define hysterical paralysis

and hysterical anæsthesia, as they have certain attributes which distinguish them from all other forms of paralysis and anæsthesia, " hysteria " cannot exist in their absence. The only possible definition of hysteria is " the condition in which hysterical symptoms are present," hysterical symptoms being in turn defined as " symptoms which result from suggestion and are curable by psychotherapy " ; *hysteria is thus a condition in which symptoms are present which have resulted from suggestion and are curable by psychotherapy.*

REFERENCES

[1] J. Babinski, *Gaz. Hebd. de Méd. et de Chir.*, p. 350, 1891; *Rev. Neurologique*, ix. 1074, 1901; *Sem. Méd.*, xxix. 3, 1909; *Exposé des Travaux Scientifiques*, p. 203, Paris, 1913.

[2] A. F. Hurst, *Seale Hayne Neurological Studies*, i. 106, 1918.

CHAPTER II

CUTANEOUS SENSIBILITY AND CUTANEOUS ANÆSTHESIA

(1) **Hysterical Anæsthesia following Hetero-suggestion.**—I have already described how Charcot believed that cutaneous anæsthesia was a stigma of hysteria, which was present before any more obvious hysterical symptoms developed and persisted after their disappearance. It had three characteristic features. Firstly, it was rarely, if ever, recognised spontaneously by the patient, and was only discovered in the course of the physician's examination. "Hemi-anæsthesia," wrote Charcot, "is a symptom, which requires to be sought for. There are many patients who are quite surprised when its existence is revealed to them." Secondly, it never caused any inconvenience to the patient, who was able to perform movements with normal accuracy so long as no paralysis was associated with it, and it never led to accidental burns or other injuries. Lastly, it always occurred in certain characteristic areas, such as one-half of the body, the whole of a limb, or the area covered by a glove or stocking (figs. 1, 2, and 3).

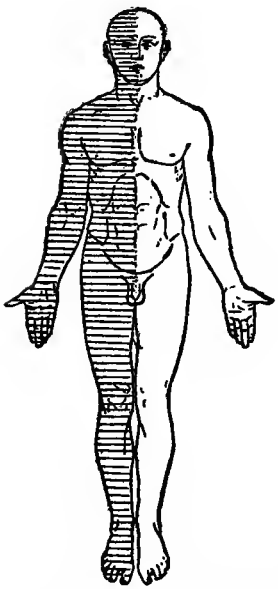

FIG. 1.—Hemi-anæsthesia in man with hysterical fits. (After Charcot.)

Doubt was first thrown on the truth of Charcot's view

by Herbert Page, who in his book on *Railway Injuries*, published in 1891, asked the question, "May not sometimes the very examination of a hemi-anæsthetic patient largely determine the hemi-anæsthesia?" And he then observed that "the examination of a patient may sometimes produce the suggestion whereby anæsthesia results."

Nine years later Babinski failed to find any disturbance

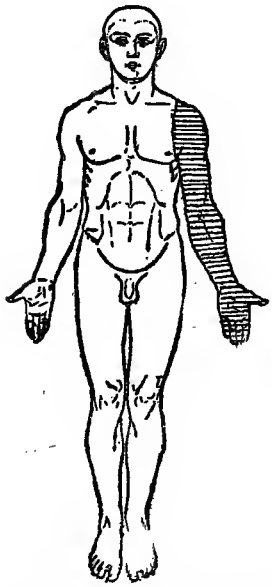

FIG. 2.—Sleeve anæsthesia in hysterical traumatic monoplegia. (After Charcot.)

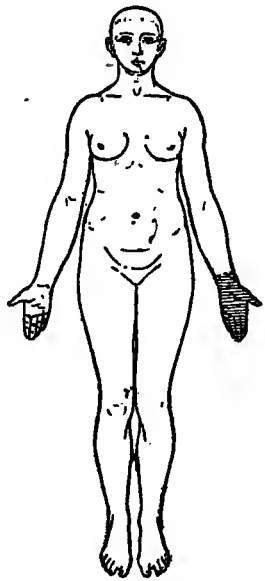

FIG. 3.—Glove anæsthesia in hysterical traumatic paralysis of hand. (After Charcot.)

in tactile, painful, thermal, muscular, and stereognosis sense in any of a hundred consecutive cases of hysterical disorders in which he was careful to avoid the possibility of suggestion. He concluded that the anæsthesia Charcot had described as so characteristic of hysteria was not recognised spontaneously by the patient, and did not cause him any inconvenience, simply because it was not present until it had been unconsciously suggested by the observer in the course of his examination.

Babinski went so far as to state that there is no such thing as hysterical anæsthesia, apart from that produced by the suggestion of the observer. In a paper on the subject published in 1908 [1] I accepted Babinski's teaching without reservation, but from time to time since then I have seen cases in which hysterical anæsthesia appeared to have developed without any possibility of hetero-suggestion by a second person. These isolated cases remained unexplained until recently, when I gradually came to recognise that certain entirely different classes of hysterical anæsthesia exist, which differ from the only form described by Charcot and the authors of most text-books of medicine and neurology by showing none of the three characteristic features mentioned above. The patient is well aware of the existence of anæsthesia before he is examined; it causes more or less inconvenience, and may lead to accidental burns and other injuries, and the areas of anæsthesia differ materially from those hitherto regarded as the only ones occurring in hysteria. The hysterical nature of these varieties of anæsthesia, which I shall presently describe in greater detail, may be extremely difficult to recognise, as the anæsthesia is always a sequel of the anæsthesia caused by some organic condition and resembles the latter in all its details.

In 1917 I carried out some investigations with Mr. R. Gainsborough on the classical forms of hysterical anæsthesia described by Charcot. Twenty-nine healthy and intelligent individuals, of whom twenty-five were medical students, who had, however, not yet worked in the wards or read any clinical medicine, were asked to pretend that they had been in a railway accident, and that they were attempting to swindle the company by claiming compensation because of paralysis of the right arm and leg, which they alleged had resulted. They were to act as well as they could the part of men trying to persuade the medical examiner that they were really suffering from paralysis.

We first asked them whether there was anything else of which they complained besides paralysis of the arm and leg. The large majority said they were quite sure there was nothing else, even after being pressed for details. These

results correspond with what occurs in patients suffering from hysterical paralysis. If they have not previously been examined, they hardly ever volunteer that they have any anæsthesia. The following leading question was then asked, " Can you feel as well on your right side as on your left ?" Twenty-two out of twenty-seven, including two who had spontaneously complained of numbness and one of coldness, said they felt less on the right side than the left. Another said he felt tingling on the right side. The remaining four said they had noticed nothing. This agrees with an observation made by Yealland, which I have since confirmed on numerous occasions. On asking a great number of non-medical individuals the following question, " Supposing you had loss of power in your wrist, fingers, and thumb, would you, or would you not, lose feeling ? " the answer he obtained was always in the affirmative. When asked what would be the limits of the loss of feeling, a line was drawn round the wrist and less frequently round the forearm, elbow, or shoulder. The replies to our leading question in the twenty-seven "malingerers" correspond with the first stage in the production of hysterical anæsthesia, the idea of which first enters the patient's head when he is asked questions on the subject.

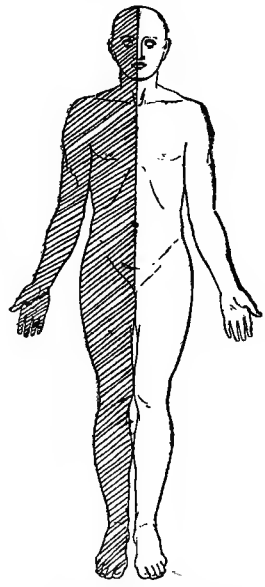

FIG. 4.—Complete hemi-anæsthesia in " experimental malingering."

Cutaneous sensibility was then roughly tested. Six of the " malingerers " had right hemi-anæsthesia (fig. 4) five had complete right hemi-anæsthesia except the face (fig. 5), twelve had anæsthesia of the whole arm and the whole leg (Fig. 6), one had hyperæsthesia of the whole arm and whole leg, and four had no anæsthesia.

Seven were asked what the area of anæsthesia would be

if only the hand and foot were paralysed: all replied that they would have anæsthesia of the distal part of the limb only, and marked out typical glove and stocking areas at various points up the wrist and forearm and ankle and leg respectively (fig. 7). I asked several other individuals what area, if any, of anæsthesia they would expect to accompany paralysis of the hand, forearm, and whole arm

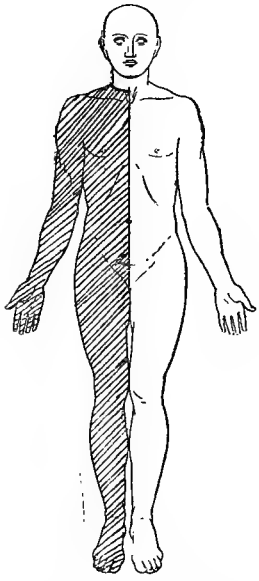

FIG. 5.—Hemi-anæsthesia with exception of face in "experimental malingering."

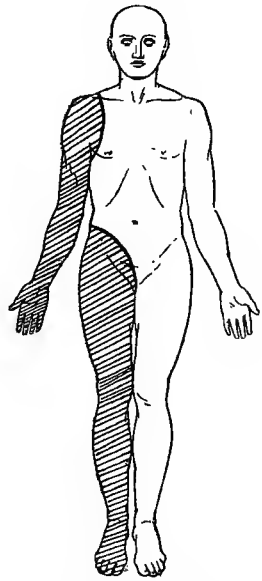

FIG. 6.—Anæsthesia of the whole arm and whole leg in "experimental malingering."

respectively. In most cases the area corresponded with the extent of the paralysis, glove anæsthesia occurring with paralysis of the hand, anæsthesia up to a line drawn round the elbow in paralysis of the forearm, and anæsthesia of the whole arm up to a line drawn through the axilla and over the shoulder with paralysis of the whole arm.

The results of these investigations correspond with the second stage in the production of hysterical anæsthesia. An investigation of cutaneous sensibility in suggestible

individuals is very likely to produce anæsthesia, especially if the way has been prepared by questions on the subject, though this is not an essential preliminary. The area of anæsthesia corresponds with the individual's own ideas, just as in these cases of experimental malingering. Figures 1 to 6 show areas which are generally regarded as typical areas of hysterical anæsthesia; they are really nothing more than the areas which anybody without knowledge of physiology would expect to become anæsthetic when the limbs are paralysed, the same individual variations being seen in these cases of "malingering" as in different cases of hysterical paralysis.

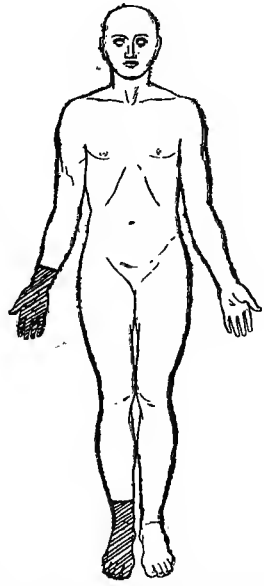

FIG. 7.—Glove and sock anæsthesia in "experimental malingering."

In all of the eleven individuals who had hemi-anæsthesia the vibration sense on the affected side of the sternum was tested, and was said to be lost. Six others were told to pretend they had hemi-anæsthesia; the vibration sense was lost in them also. Three medical men, who were asked whether they would lose the vibration sensation on the right side of the sternum if they had hemi-anæsthesia, also answered in the affirmative. This corresponds with the well-known sign, which has sometimes been regarded as evidence of hysteria, sometimes of malingering, but is really common to both. It is what would be expected to occur unless the question was carefully thought out, when it would be realised that the sternum vibrates as a whole, so that the vibrations produced by the tuning-fork would be felt with the normal half of the sternum whether it was placed to the right or the left of the middle line.

(2) **Hysterical Anæsthesia following Stupor.**—A condition of stupor was not uncommon in soldiers, who were exposed

to some exceptional emotional disturbance, when their resisting power had already become lowered by prolonged exposure to terrifying experiences, especially if they were suffering from great physical fatigue, the result of strenuous exertion, want of sleep, and perhaps insufficient food. In this condition the patient's mind appears to be so absorbed with his own innermost thoughts that he gives no attention to the external world. He appears to see, hear, and feel nothing. He does not blink when a stick is waved in front of his face, nor jump when a loud noise is made just behind him. He pays no attention to tickling or to painful stimuli applied to his skin. Sooner or later spontaneous recovery occurred in most cases. After a period of confusion the patient became normal in every way, and his vision, hearing, and sensibility to all forms of cutaneous stimuli returned. In most cases the intermediate period of confusion was very short, but it was sometimes prolonged. If during this period the patient happened to be under a medical officer, whose clinical enthusiasm prompted him to make repeated examinations of his motor and sensory functions, the patient's dawning interest in the outside world was focussed on these functions, some deficiencies in which thus became perpetuated as hysterical symptoms. A very striking example of this was a patient who happened to be the subject of intense study by a number of neurologists in the early days of the war, when the true hysterical nature of so-called "shell-shock" was not fully understood. The sensory deficiencies natural to a condition of profound stupor were consequently perpetuated after the stupor disappeared, and resulted in the most complete and widespread hysterical loss of all forms of sensation, except hearing and seeing, which I have ever observed.

Total amnesia with hysterical paralysis, contractures, analgesia and mutism due to emotional strain; recovery of memory after twenty-two months, and from paralysis after twenty-eight months.—Private M., aged 23, with no personal or family history of neuroses, was slightly wounded in the thigh in May 1915, after serving from the beginning of the war. He returned to the front in October 1915. He

was quite fit until February 19, 1916, when he had to be forcibly prevented from going over the parapet to attack some German mortars which were firing at his trench. He then became dazed, and on reaching the aid post he could not answer questions, but he obeyed simple commands, such as to put out his tongue. He believed he was still in the trenches, which were being heavily shelled ; his eyes were fixed on imaginary trench-mortar shells coming towards him. His knee-jerks were brisk, there was an extreme degree of pseudo-ankle clonus, but the plantar reflexes were flexor. The bowels and bladder remained under control. Two days later the knees and ankles had become stiff ; the legs, hands, and face were anæsthetic and analgesic. When he reached England on March 2, his expression was apprehensive, and he started at every sound, both when awake and asleep. In his dreams he saw the ghosts of Germans he had bayoneted come to take revenge on him, and he heard them fire at him. He was still unable to speak, but he answered questions by nods and signs and in writing. He was able to walk with assistance. He was treated by hypnotism, and the hallucinations disappeared, but his physical and mental condition rapidly deteriorated.

I saw him for the first time in December 1916, eleven months after the onset. He could not speak, and all four limbs were now completely paralysed, except that he was able with a great effort to make slight movements at his left elbow joint. An extreme degree of contracture was present : the legs were rigidly extended with the feet plantar flexed ; the arms were extended and the fingers tightly clenched, though the metacarpo-phalangeal joints were extended. It was almost impossible to produce any passive movements, but the contractures were entirely hysterical, as they relaxed completely under an anæsthetic and during sleep. Total anæsthesia and analgesia of the whole body, including the conjunctiva, cornea, and buccal mucous membrane, were present, except that passive movements at the elbow were painful, and he occasionally suffered from toothache. The anæsthesia disappeared, at any rate to some extent, during sleep. Although deep pressure over

the abdomen produced no sensation, the sensibility of the bladder and rectum appeared to be present, as he retained perfect control over them. The sense of taste and the sense of smell were obviously much impaired and the fields of vision seemed to be greatly contracted, but hearing was abnormally sensitive, and the auditory-motor reflex was extremely brisk. The conjunctival, corneal, and all skin reflexes were absent. When the contractures relaxed under partial anæsthesia, the deep reflexes were found to be normal.

On December 15, 1916, vigorous suggestion with the aid of an intralaryngeal electrode during light etherisation restored the power of whispering. It was then found that the patient had total loss of memory; he had no idea who or what he was, he did not realise that his anæsthetic legs belonged to him, and he had no knowledge of the meaning of words.

During the following months he learnt to talk a kind of pidgin-English, but the meaning of every word had to be taught, and he used each word in his limited vocabulary for a variety of meanings. All liquids were "tea," and when petrol was poured into the tank of a motor-car he was in, he called out "table has tea," table being for some obscure reason the name he applied to all vehicles. "Hand" represented a hand and a glove, and "to hand" was to hit. A word taught by other patients in fun would never be given up, so that all forms of meat, chicken, and fish were called "puss." His only numbers were one and six, which represented anything more than one, except a very large number, which was sixty-six, or a still larger number, which was six-sixty-six. The sight of his own face in a mirror always terrified him; he did not realise who it was he saw, and turned his head away, saying, "No like you, chick." On seeing in the mirror the face of the sister, who was standing by his side, he was greatly amused, and said, "See six sisters." All attempts to teach ideas of time, space, and colour failed, and he did not recognise any of his relations, even when his father was brought to him in the middle of the night, in the hope that he might know him at the

moment of waking. He remembered recent events, and called people by names which he invented himself: a bald patient was "no-hair chick," and men who limped badly were "no-leg chick" and "six-leg chick"; all officers in uniform were "Major" and civilians "Mister," or, if friends, "Mister Chick."

In spite of treatment no improvement in the condition of his limbs had occurred by October, 1917, though he was able to sit up in a chair and enjoyed being taken out of doors. He delighted in childish toys, and in a general way his mind was that of a year-old child. He was quite happy, but he was becoming very emaciated; it was difficult to persuade him to eat, as he had completely lost the senses of taste and smell, and he apparently never felt hungry.

On November 22, 1917, for no obvious reason, he had a headache, and became excited in the evening. His memory began to return during the night, and he talked incessantly. The next day he realised the deficiencies in his speech, and wished to have them corrected. When told a word, he repeated it correctly and remembered it, and he began to form proper sentences. On November 24 I cured a man suffering from hysterical aphonia with a laryngeal sound in his presence. Though this had failed on many occasions since he learnt to whisper nearly a year before, it now cured him instantaneously, to his intense delight. He felt something snap in his head, and immediately afterwards he talked quite normally, and the memory of his home and his past life flowed back. His father came the next day, and he knew him at once. He soon remembered his experiences in France, but his life in hospital for twenty-one months was a blank, as it seemed to him that he was in France only a few days ago. He had a vague recollection of very recent events, and he knew the men in the ward, but did not remember friends who had gone out only a week before. He remembered "feeling funny with a buzzing head," then "something in his head was suddenly relieved," and the buzzing stopped when his memory returned.

Complete anæsthesia and analgesia were still present, and the cutaneous, conjunctival, and corneal reflexes were

absent. With his eyes bandaged, the only difference he could recognise between hot and cold water, tea and beer, was that the tea was " sweet " and the beer was " bitter." Salt was recognised as a powder, but without taste. Jelly was recognised, as it dissolved so quickly in the mouth, but butter was also taken to be jelly. Bread was indistinguishable from fish, and he could not recognise any difference whether it was spread with butter, mustard, or salt. Marmalade appeared sweet, but was not recognised ; he guessed that chutney was apple.

His mental condition was now perfectly normal, but for some time very little improvement occurred in the condition of his limbs. With re-education the left arm slowly improved, but it was not until he was transferred to the Seale Hayne Hospital in April 1918 that any marked change occurred. At the end of a fortnight his right arm, which had hitherto remained rigid and paralysed, had improved so much that he could write long letters, brush his hair, and feed himself, but all movements were stiff, shaky, and slow. The left arm improved at the same time, but there was still no recovery of voluntary power in the legs. On May 1 a renewed attempt was made to overcome the rigidity of his legs. This was so far successful that slight voluntary power returned in the feet. Steady improvement followed, until all movements became possible, the adductor spasm of the thighs being the last to relax. By May 31 he could stand with very little assistance, and could perform all ordinary movements with his arms, though some rigidity was still present. On June 2, his twenty-fifth birthday, he stood without support, and after being helped for a few minutes, he walked without assistance round the quadrangle, after having been paraplegic for twenty-eight months. His physical condition now greatly improved, and by June 20 he was able to take charge of the basket-making shop. The sensibility of his skin and mucous membrane slowly returned without special treatment, but was still somewhat deficient in August. The superficial reflexes returned *pari passu*. Thus the abdominal reflexes were absent so long as anæsthesia was complete. When at

length strong faradic stimulation could just be felt on the left side, a weak reflex appeared on this side. The right reflex only returned at a later date, when the right side of the abdomen was no longer completely anæsthetic, but it was still weaker than the left, corresponding with the fact that sensibility was more nearly normal on the left than on the right side. In the same way the corneal and conjunctival reflexes returned with the return of the sensibility of the cornea and conjunctiva.

By September 1918 recovery was complete. The last trace of contracture of the fingers and of unsteadiness in gait had disappeared. The patient had regained his weight, and was in every way as fit as when he first joined the army. He wrote to me at Christmas, 1919, saying that he had been back at work for over a year and remained perfectly well.

(3) **Hysterical Anæsthesia following the Anæsthesia caused by Injury to Peripheral Nerves.**—The paralysis and anæsthesia following an injury to a peripheral nerve, which are caused by the interruption in the nervous impulses passing between the central nervous system and the periphery, may both be perpetuated as hysterical symptoms when the interruption is no longer present.

A gunshot wound in the immediate neighbourhood of a nerve produces minute changes in its structure, which quickly disappear, but, evanescent as these concussion changes are, they are none the less organic, and the paralysis and anæsthesia they produce are primarily of organic origin. The patient may at first make repeated attempts to contract the muscles supplied by the nerve, but as he invariably fails, he finally discontinues, and realises that the muscles are paralysed. If the true nature of the condition is recognised and the patient is encouraged to repeat his attempts every day, he will find in a very short time that his power is returning. If, on the other hand, the condition is misunderstood, and the patient is ordered treatment with electricity and massage, and is given to understand that he may have to continue with this treatment for weeks or months before recovery can take place, the original tendency to perpetuate the incapacity by auto-suggestion is greatly

strengthened by the hetero-suggestion involved in the treatment, with the result that by the time the nerve has completely recovered, the organic incapacity is replaced by an exactly similar hysterical incapacity. In the same way the primary organic anæsthesia is perpetuated as hysterical anæsthesia with all its characteristics unaltered, particularly if the patient's attention has been drawn to its extent and its exact nature by the thorough investigations of a keen and interested medical officer in the early stages, when the condition was still organic.

If the nerve is more seriously damaged, especially if it has been divided and subsequently sutured, the primary organic condition lasts for a longer period, and its characteristics are all the more likely to become vividly stamped on the patient's mind, and to become perpetuated as hysterical phenomena, when recovery from the actual injury at last occurs.

I am convinced that the voluntary power of paralysed muscles returns before the response to faradism when an injured or divided and sutured nerve is regenerating—an opinion which is shared by most observers. When complete paralysis has been present for many weeks, it is natural that a man should cease to make an effort to move the affected part, so that it is extremely likely that he will not become aware of his returning power at the first moment that the regenerating nerve is capable of conveying motor impulses. If left to himself, he will probably only make the discovery some weeks later, when a considerable degree of power has returned and the normal electrical reactions are re-established. If he is receiving regular electrical treatment, the discovery of a response to faradism is likely to prompt the operator to tell him to make an effort to move, which will in all probability be successful, owing to the suggestive effect of the ocular demonstration of the movement produced by the electricity. Under more favourable conditions the patient is seen frequently by the physician, who encourages him to make daily efforts to contract the paralysed muscles, with the result that the returning power is recognised at an early stage—frequently before there is

any response to faradism. I believe that the exceptionally early recovery of function after nerve suture, which is occasionally observed, is due to the patient having discovered that he can contract the paralysed muscles within the first few days after the nerve has regenerated sufficiently for a few feeble impulses to be conveyed along it.

If from the beginning no attention is paid to the anæsthesia, it disappears spontaneously when the motor symptoms are cured. If, however, it has been carefully investigated, and its extent and nature have become thoroughly recognised by the patient, it is likely to persist as hysterical anæsthesia. It can then be cured almost instantaneously by the suggestive effect of the application of a faradic current, the patient being first told that his sensation will at once return when the electricity is applied. He quickly feels the electricity, and directly afterwards it is demonstrated to him that he can now also feel the slightest touch as well as pain, and can distinguish accurately between hot and cold. This immediate recovery with suggestion proves that the condition must be genuinely hysterical.

The exact area involved when the condition is no longer organic depends largely upon the intelligence of the patient and the interest he takes in the examination of his anæsthesia, and partly on the physiological and anatomical knowledge of the observer, who would be likely to influence the patient by his own ideas on the subject, both whilst the condition was organic and after it became hysterical, unless he was unusually expert in avoiding the liability to suggest what he expected to find. The following three cases are selected from several we observed in which hysterical anæsthesia showed a distribution exactly similar to that produced by an organic injury to a peripheral nerve.

Hysterical anæsthesia of the median nerve following a wound of the forearm.—Private B., aged 36, was wounded in the right forearm on September 5, 1918. His hand became paralysed, and there was anæsthesia in the area of the skin supplied by the median nerve. After being treated by massage for three months, he was admitted to Seale Hayne Hospital, under Captain S. H. Wilkinson, on January

20, 1919. Hysterical contracture of his fingers prevented him from flexing them, and he complained of numbness of the thumb and the first two and a half fingers (fig. 8). He was treated by persuasion and re-education, and at the end of two days the motor power of his hand was normal. Sensation was then restored in a few minutes by suggestion with the aid of faradism.

Hysterical anæsthesia of the cutaneous branch of the musculo-cutaneous nerve following cellulitis of the upper arm.—Rifleman T., aged 20, was slightly wounded on September 5, 1918, and was given antitetanic serum under the skin of the left upper arm. Following this, he developed acute cellulitis, for which two incisions were made. He was sent to a hospital in England three weeks later, his arm being immobilised on a splint. Hysterical monoplegia of the

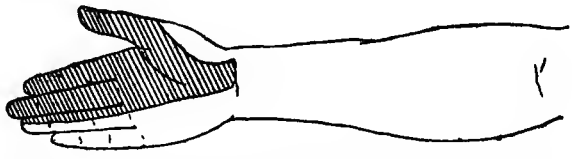

FIG. 8.—Hysterical anæsthesia in median nerve area.

left arm developed, and for three months he was treated with electricity and massage. He was transferred to Seale Hayne Hospital, under Captain S. H. Wilkinson, on January 28, 1919, complaining of inability to close his hand and anæsthesia in an area which corresponded exactly with that supplied by the cutaneous branch of the musculo-cutaneous nerve (fig. 9), which had presumably been inflamed as a result of the cellulitis. The paralysis was probably hysterical from the start, and was rapidly cured by persuasion, sensation being restored in a few minutes by means of suggestion with the aid of faradism.

Hysterical anæsthesia of the ulnar nerve following a wound of the forearm.—Shoeing-smith A., aged 22, was wounded on the inner side of the right forearm on October 2, 1917. He was unable to flex his fingers, and completely lost the sensation of the little and the inner half of the ring fingers,

and the front and the back of the inner side of the palm of the hand. He was sent to hospital in England on October 18, 1917, and on February 28, 1918, he had an operation on the ulnar nerve. No notes are available except that "the operation was specifically for the ulnar nerve." No improvement followed, and he was treated by massage and

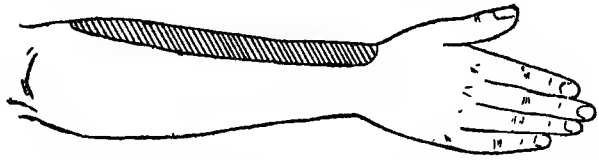

FIG. 9.—Hysterical anæsthesia in musculo-cutaneous area.

electricity for ten months. He was admitted to Seale Hayne Hospital, under Captain S. H. Wilkinson, on February 16, 1919, with inability to move the ring and little fingers, which were held in a semi-flexed position, and there was anæsthesia in the area of the skin supplied by the ulnar nerve (fig. 10). By persuasion and re-education for half an hour the motor power was completely restored, and sensa-

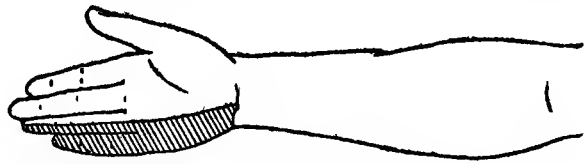

FIG. 10.—Hysterical anæsthesia in ulnar nerve area.

tion returned in a few minutes as a result of suggestion with the aid of faradism.

Direct treatment of hysterical anæsthesia following a nerve injury is, however, often unnecessary, even if the patient is well aware of its existence; it is sufficient to point out to him, as in the following case, that as soon as his power of movement returns, the loss of sensation will disappear.

Spontaneous disappearance of hysterical anæsthesia of the ulnar nerve on recovery from associated paralysis.—Private

B. was wounded in the upper arm on August 29, 1918. He developed a contracture of the ring and little fingers, and cutaneous anæsthesia of the area of the skin supplied by the ulnar nerve. He was transferred to Seale Hayne Hospital, under Major J. F. Venables, on November 10, 1918, with the ring and little fingers firmly flexed into the palm of the hand, and with cutaneous anæsthesia in the area supplied by the ulnar nerve. The paralysis and contracture were treated by persuasion and re-education, and it was explained to him that as soon as he could move his fingers normally the numbness would go. In half an hour he was completely cured, the anæsthesia as well as the contracture having disappeared without any direct treatment by faradism or other form of suggestion.

In the following two cases the anæsthesia, though hysterical, was so profound that the patient injured himself without being aware of it at the time.

Hysterical anœsthesia of the median nerve, resulting in accidental injuries to the anœsthetic area.—Private I., aged 19, was wounded on October 11, 1918, in the left forearm. His arm and hand were put on a splint, and he was transferred to England a week later. On October 20 he tried to use a fork for the first time, and discovered that he could not feel it. He told his medical officer, who found that he had anæsthesia in the area supplied by the median nerve. He was transferred to Seale Hayne Hospital, under Captain S. H. Wilkinson, on February 2, 1919, suffering from hysterical paralysis and contracture of the fingers,

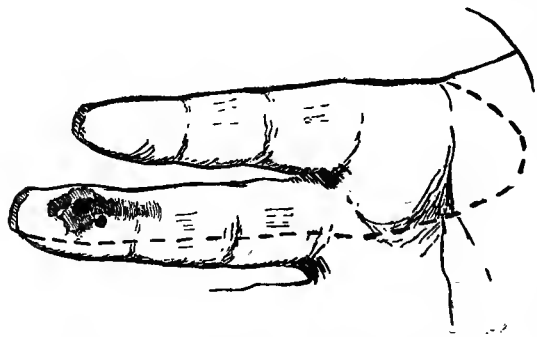

Fig. 11.—Hysterical anæsthesia in median nerve distribution, with unfelt accidental burn.

FIG. 12.—Burn over first and middle finger in hysterical anæsthesia area.

which were fixed in a semi-flexed position. The margin of the outer side of the nail of the middle finger was ulcerated over an area the size of a shilling, and on the extensor aspect of the first finger there was another superficial ulcer (fig. 11). He stated that two days previously he had tried to get the feeling back in his fingers and thumb by holding them in front of the fire. He had been unable to do so, and had not been aware that he had burnt himself until the following morning, when he found large blisters had developed on his fingers.

On the morning of admission his hand was treated by persuasion and re-education, and at the end of half an hour he could open and close it quite strongly in a normal manner. The anæsthesia was treated by suggestion with the aid of faradism, and in a few minutes his cutaneous sensibility was completely restored. He had no further trouble with his hand, the ulcers rapidly healing, and a fortnight later he was discharged.

Hysterical anæsthesia of the median nerve, resulting in an accidental injury in the anæsthetic area.—Private L., aged 36, received a penetrating wound of the right forearm on October 4, 1918. This was followed by loss of muscular power of the hand and anæsthesia of the area of skin supplied by the median nerve. The wound was excised at the C.C.S. He was admitted to Seale Hayne Hospital, under Captain A. Robin, on February 20, 1919, with hysterical contracture of the fingers, the forefinger being rigidly extended and the hand blue and cold. He had anæsthesia in the area of skin supplied by the median nerve, and on the outer side of the second finger there was an ulcerated area about the size of a shilling, which he stated was caused by his trying to hold a lighted match a few days previously (fig. 12). The contracture was cured by re-education and persuasion, and the anæsthesia disappeared completely within a minute as a result of suggestion by means of faradism. With the restoration of the circulation and disappearance of anæsthesia the ulcer rapidly healed.

(4) **Hysterical Anæsthesia following Cerebral and Spinal Lesions.**—The anæsthesia associated with the paralysis

caused by disease or injury of the brain and spinal cord may be perpetuated as an hysterical symptom after partial or complete recovery from the organic lesion, in the same way as the anæsthesia associated with the paralysis caused by injury to peripheral nerves. I have not, however, made many accurate observations on this subject, except in cases of spinal concussion in which total paraplegia followed a blow on the back. In such cases signs of organic paraplegia were often present at first, and anæsthesia was frequently discovered up to the level of the spinal segment which was principally involved in the injury. Both paralysis and anæsthesia sometimes persisted for months or even years after the organic lesion had so far recovered that it could no longer be responsible for any disturbances in the spinal functions. The paraplegia was almost always amenable to the rapid form of psychotherapy, in which explanation, persuasion, and re-education resulted in more or less complete recovery in the course of a single treatment lasting perhaps an hour. If no attention was paid to the accompanying anæsthesia at the time, it would always be found to have disappeared with the paralysis when a careful examination was made on some subsequent occasion. If, however, the sensory functions were investigated during the treatment, they would be likely to persist and require further psychotherapy before they finally disappeared. A patient recovering from paralysis, which was the only symptom in which he was really interested, as it was the sole cause of his incapacity, would subconsciously expect any associated but to him unimportant symptom, such as anæsthesia, to disappear when the paralysis disappeared; but if his attention was directed to his anæsthesia immediately before or during the treatment, he might very well expect that a symptom which so greatly interested his physician would require separate treatment and he would not recover spontaneously.

(5) **Hysterical Anæsthesia following the Anæsthesia caused by Peripheral Anæmia.**—(*a*) *Peripheral anæmia following disuse and vasomotor spasm.*—Whenever the hands or feet of healthy individuals get abnormally cold, the anæmia resulting from the contraction of the peripheral

vessels is accompanied by a feeling of numbness and diminution in the acuteness of cutaneous sensibility. Individuals with a feeble circulation are particularly apt to get numb extremities in cold weather, and in Raynaud's disease a similar but much more severe anæmia with a greater deficiency of cutaneous sensibility occurs as a result of vasomotor spasm, even when the weather is not unusually cold.

A man with a poor circulation knows by experience that the best means for preventing attacks of peripheral anæmia is exercise, and that when his hands are becoming abnormally cold and numb, active movements may restore the circulation and cause the numbness to disappear.

It is natural that the immobility caused by paralysis or contracture of a limb, whether the latter is organic or hysterical, should result in deficient circulation, accompanied by numbness and anæsthesia. As the deficient circulation is continuous instead of intermittent, as in the conditions already referred to, the anæsthesia becomes more profound, especially in individuals who have a feeble circulation. The anæsthesia may be so severe that the patient may burn himself without being aware of it, as in the case described below. When the circulation is temporarily improved by immersing the limb in hot water, normal cutaneous sensibility returns, and in hysterical cases the improved circulation, which results from the recovery of mobility and disappearance of spasm under psychotherapy, is immediately followed by a return of normal cutaneous sensibility, even if the condition has persisted for months or as long as two or three years.

If, however, much attention has been paid to the anæsthesia, which results from the poor circulation in hysterical paralysis and contracture, and its exact extent has been carefully mapped out on several occasions, the symptom takes such a prominent place in the patient's mind, instead of being one of which he is hardly aware, that it is very liable to be perpetuated by auto-suggestion when recovery from the paralysis and contracture takes place. The patient does not associate the anæsthesia with the diminished circulation, but regards it as an independent symptom,

which will require separate treatment by re-education or suggestion after the paralysis and contracture have been dealt with. That the anæsthesia is purely hysterical when it persists after recovery from the paralysis is proved by its immediate disappearance with psychotherapy, although this has no effect so long as the paralysis is still present, as the anæsthesia is then a direct result of the peripheral anæmia, and is incapable of amelioration so long as the latter persists.

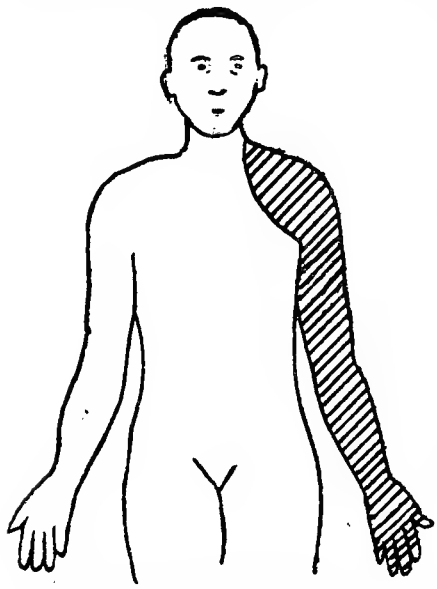

FIG. 13.—Hysterical anæsthesia of arm following peripheral anæmia.

Hysterical anœsthesia following the anœsthesia caused by peripheral anœmia associated with hysterical paralysis of the hand.—Sergeant H., aged 23, underwent an operation on January 18, 1918, for the removal of a ganglion from the dorsum of his left hand. He was discharged to duty six weeks later, but two days after returning to work the hand became blue and swollen, and he was readmitted to hospital. The swelling was lanced, a quantity of pus evacuated, and a splint and fomentations were applied. The wound had healed four weeks later, but the splint was kept on another fortnight. The patient then found that he could not move his fingers, and that his whole arm was flaccid and useless. He was sent to the electrical department, and a few days later, while smoking a cigarette and holding it carelessly in his right hand, he brought the burning end close to his left hand. A comrade told him to be careful, but not before he had burnt his finger and caused a blister to appear. He felt no pain from the burn nor from

the blister, and this was the first time that it had occurred to him that his hand was anæsthetic. He called the doctor's attention to it: the arm was examined and found to be somewhat wasted, but the anæsthesia was not investigated. A month later the patient was transferred to another hospital, and here the anæsthesia was tested for the first time, and was found to extend as far upwards as the neck (fig. 13).

On admission to Seale Hayne Hospital, under Captain W. R. Reynell, on May 19, 1918, complete flaccid monoplegia of the left arm was present, and the hand was blue, cold, and œdematous. Complete anæsthesia and analgesia were present over the area, shown in fig. 12. Treatment by explanation followed by vigorous persuasion and re-education was sufficient to restore the power of the muscles of the shoulder and upper arm in about ten minutes; but there still remained a very slight degree of wrist-drop, and the fingers and thumb could not be moved. After a second treatment, lasting about half an hour, the power of the fingers was so far restored that the grip on the dynamometer registered 110, and the blueness and swelling had completely disappeared. Two days later the grip was 140, and the patient took part in boxing practice. A fortnight after admission the grip was 180, which is above normal for the left hand, the grip with the right hand being 240. The anæsthesia, which had not altered in degree or extent, was now treated by direct suggestion with the aid of faradism; normal sensibility of the whole arm was restored in a few minutes.

It is clear that in this case the primary anæsthesia which led to the accidental burn was not hysterical. It was caused by the peripheral anæmia, with which the disuse resulting from the hysterical paralysis was associated. When the extent of the anæsthesia was investigated, the examination resulted in its spread by hetero-suggestion, a large hysterical element being now present, as the peripheral anæmia could only account for anæsthesia of the hand and possibly of the forearm. The restoration of the circulation which followed the cure of the paralysis must have caused the anæsthesia due to the peripheral anæmia

to disappear; but as most of the anæsthesia was already hysterical, this part also became perpetuated by auto-suggestion, when the original cause was no longer operative. Consequently the whole of the anæsthesia was ultimately

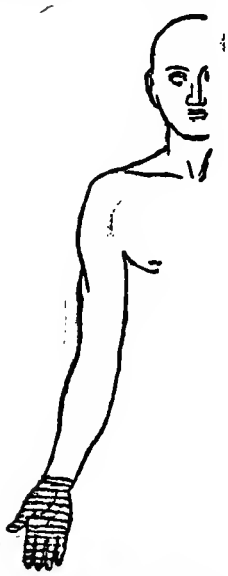

FIG. 14.—Anæsthesia in ischæmic myositis following tight bandaging. (After Purves Stewart.)

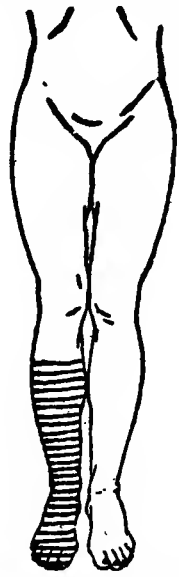

FIG. 15.—Anæsthesia in ischæmic myositis following infective thrombosis of external iliac artery. (After Purves Stewart.)

hysterical, and, being hysterical, was capable of being cured by psychotherapy.

In the rare cases in which the anæsthesia associated with hysterical paralysis and contracture appears to develop spontaneously in the absence of any medical examination, it is probably in the first instance due, as in the case just described, to the associated peripheral anæmia. It is thus not primarily hysterical, being only hysterical if it persists after recovery from the paralysis and contracture.

(*b*) *Peripheral anæmia following obstruction to large blood-*

vessels.—Injury to one of the large blood-vessels of a limb results in disturbances of sensation. Mme. Athanassio-Bénisty in 1916 described the sensory disturbances in these cases as consisting of complete anæsthesia, which extends over an irregular area bearing no relation to the distribution of any peripheral nerve. It sometimes involves only the tips of some or all of the fingers, and in other cases it extends over the whole hand or foot. The area corresponds with that in which the vasomotor and trophic changes are most marked. More recently Purves Stewart, in a description of the ischæmic myositis resulting from tight bandaging, ligature of an artery, and spontaneous obstruction of an artery by thrombosis, gave a similar account of the associated anæsthesia. As he found that the area involved never corresponds with that supplied by a peripheral nerve, he concluded that it could not be due to direct pressure on a nerve or interference with the blood supply of a nerve. It is clearly of exactly the same nature as the anæsthesia already described as resulting from the peripheral anæmia due to cold in normal individuals, vasomotor spasm in Raynaud's disease, and disuse in paralysis of a limb. Whatever the exact explanation of the anæsthesia may be, there is a great tendency for it to be perpetuated as hysterical anæsthesia after the original cause has disappeared. The glove and stocking areas shown in some of Purves Stewart's cases (figs. 14 and 15) are identical with those produced by direct suggestion, and although, of course, the origin is not in these cases primarily due to suggestion, the exact definition of the anæsthesia corresponds with what the patient himself would expect, and may perhaps be in part hysterical from the moment it is mapped out. In the few cases of this kind I have seen in which the anæsthesia persisted after the disturbance of circulation had diminished or disappeared, it was readily cured by re-education or suggestion.

REFERENCE

[1] A. F. Hurst and S. H. Wilkinson: *Seale Hayne Neurological Studies*, i. 171, 1919.

CHAPTER III

PATHOGENESIS OF HYSTERICAL CUTANEOUS ANÆSTHESIA

LIKE all other hysterical symptoms, each of the five classes of hysterical anæsthesia which I described in my first lecture is the result of suggestion. In the first class, the classical form which has alone been recognised in the past, the idea that a certain area of skin is insensitive to tactile, thermal, and painful stimuli is in the first instance unconsciously suggested to the patient by the physician who examines him. The second class, the anæsthesia which may follow a condition of stupor, is also due to hetero-suggestion, although it is preceded by anæsthesia which is neither due to organic disease nor to suggestion, but is the result of profound inattention during the stuporose period. In the remaining classes the anæsthesia is primarily organic in origin, being caused by disease or injury of the brain, spinal cord, or peripheral nerves, or by peripheral anæmia. It makes so great an impression on the patient's mind that it is perpetuated as an hysterical symptom after the organic cause has disappeared. This is no doubt in part a result of auto-suggestion, but it is clear that such auto-suggestion would very rarely occur if not prompted by a certain amount of unconscious hetero-suggestion on the part of the physician, whose interest in the anæsthesia attracts the patient's attention to what would otherwise be to a great extent ignored, owing to the small inconvenience the anæsthesia causes compared with the associated paralysis. So impressed was Babinski with the part taken by the observer in suggesting hysterical anæsthesia that he expressed his belief that the condition never occurred in the absence of such suggestion. Whilst I agree with him so far as the first

class of hysterical anæsthesia is concerned, I have certainly seen cases of the third class—that caused by injury to peripheral nerves, and possibly in the fourth and fifth classes, in which any such hetero-suggestion could be excluded with certainty. Although in most of these cases hetero-suggestion undoubtedly plays the predominating part, auto-suggestion generally occurs as well, and in rare cases it is alone responsible for the production of the anæsthesia.

The next question to discuss is by what process anæsthesia develops when it has once been suggested by hetero-suggestion, auto-suggestion, or the two combined. I believe that a consideration of the second class of hysterical anæsthesia, that following stupor, affords the only satisfactory explanation. In order to feel, one has to pay attention. If the whole mind is absorbed with one's thoughts, tactile and other cutaneous stimuli will not be felt. Most healthy people are aware of this from their own experience. If one is intensely interested in the occupation in which one is engaged, a light touch will not be perceived, and the discomfort caused by external heat or cold or by some form of cutaneous irritation will not be felt until the mind is once more free to attend to such mundane affairs. The importance of this conception can only be fully realised by those who have had the opportunity of examining cases of stupor, such as I have described as occurring in the overwrought soldier. The patient's mind was obviously absorbed in the contemplation of his own thoughts, which were generally painful in character, and were often so vivid that they could be correctly described as visual or auditory hallucinations. Tickling the face or the soles of the feet, pinching or pricking the most sensitive parts of the skin, or the application of very hot or very cold objects failed to elicit the slightest sign indicating that the individual had felt anything. The conjunctiva, cornea, and nasal mucous membrane were equally insensitive, and all superficial reflexes were abolished. Inattention had resulted in anæsthesia.

When an individual accepts the suggestion that he cannot

feel over a certain area of skin, he withdraws his attention from this area, and he consequently no longer feels any stimuli applied to it. This conception will be more fully discussed later, when I shall show how hysterical blindness may result from not looking, as without looking it is impossible to see, and how hysterical deafness may result from not listening, as without listening it is impossible to hear. For feeling, seeing, and hearing are active processes, and require an effort of the will just as much as walking. The latter becomes automatic in process of time, but if an individual accepts the suggestion that he cannot walk, he no longer makes the active effort which is required. Without this no walking can occur, however automatic the movement may appear to be in a healthy individual. In the same way, if an individual accepts the suggestion that he cannot feel, see, or hear, he ceases to make the active effort involved in the process of attention to cutaneous, visual, or auditory impulses. To a normal man, feeling, seeing, and hearing appear to be the result of cutaneous, visual, and auditory stimuli, respectively acting upon the skin, eyes, and ears, a point of view which was expressed by Wordsworth :—

> "The eye—it cannot choose but see :
> We cannot bid the ear be still ;
> Our bodies feel, where'er they be,
> Against or with our will."

But this is, in fact, only true if the individual is actively attending, although this active attention is as little a conscious process as the performance of the individual movements in walking.

A study of the behaviour of the cutaneous reflexes in hysteria will show that an anatomical basis for hysterical anæsthesia must exist, which depends upon the removal of the structural foundation of the psychological process of attention. A reflex is the automatic response to a peripheral stimulus and is entirely independent of consciousness. It is thus inconceivable that a reflex could be abolished unless some alteration had occurred in the

structures which control the reflex arc. The abolition of a cutaneous reflex in organic disease is, for example, regarded as a proof of the existence of a lesion interfering with these structures. Apart from the abolition of the plantar reflex when the feet have become cold owing to the feeble circulation caused by disuse in hysterical paraplegia, superficial reflexes are only diminished in activity or entirely abolished in hysteria in the rare cases of hysterical anæsthesia in which sensibility is almost or completely absent. In these exceptional cases of total anæsthesia the corresponding superficial reflexes are lost, just as all superficial reflexes are lost in the total anæsthesia present in severe stupor. Recovery from total hysterical anæsthesia, either spontaneously or as a result of treatment, is accompanied by a return of the superficial reflexes, just as they return when a condition of stupor passes away.

The simplest explanation of the behaviour of the superficial reflexes in hysterical anæsthesia is that the structural basis of the psychological act of attention consists in some change which leads to a diminution in the resistance offered at each synapsis of the sensory tract. Perhaps this is in the nature of a throwing out of dendrites, or it may depend upon some biochemical change in the material which occupies the space between the dendritic terminations of adjacent neurones. Whatever it may be, the increased resistance which is present when attention is very deficient results in anæsthesia, and at the same time a block is produced in the reflex arc which results in diminution or abolition of the reflex.

The usual method of treating hysterical anæsthesia by means of a strong faradic current depends upon the fact that the anæsthesia, however severe, is very rarely absolute. A very powerful stimulus, such as that afforded by a strong faradic current applied with a wire brush, can break through the increased resistance at the synapses in the sensory tract in spite of the almost complete inattention which is present; it is consequently felt. Directly this occurs the patient's dormant attention is awakened: with a little persuasion, and perhaps some explanatory conversation,

which may be looked upon as acting, at any rate in part, as a counter-suggestion to the original suggestion of anæsthesia, the patient is quickly brought to attend to the stimuli affecting the previously anæsthetic areas of his skin, and normal sensibility returns.

If the anæsthesia is absolute, this method cannot be used, as, however strong the painful stimulus may be, it will not be felt. In such a case it may be suggested to the patient that the faradisation of the skin at the boundary of the anæsthetic area will lead to the progressive retraction of this area. The most satisfactory method, however, is to use no suggestion at all, but to explain to the patient why he cannot feel—that he has become accustomed to pay no attention to stimuli affecting the anæsthetic area, and that if he will concentrate his attention on this area sensation will return. When he understands what he has to do, recovery of normal sensibility at once returns, and with the return of normal attention the abnormal resistance at the synapses disappears and the abolished cutaneous reflexes reappear.

CHAPTER IV

THE SUPERFICIAL REFLEXES IN HYSTERIA

THERE has been much controversy about the behaviour of the superficial reflexes in hysteria. Differences of opinion are in part due to insufficient knowledge as to the normal variations in the reflex under different physical conditions. My investigations on these subjects have been made principally in connection with the abdominal reflex, but I shall also refer to some observations on the plantar reflex.

The Abdominal Reflex.—Authorities differ as to the frequency with which the abdominal reflex may be absent in normal individuals. A recent investigation by Söderbergh [1] on 700 normal individuals between the ages of 2 and 68 shows, however, that in favourable conditions it can almost invariably be obtained. It is important to adopt a proper method of examination before concluding that it is absent. It is particularly difficult to detect in individuals with much subcutaneous fat or flabby abdominal muscles. It is necessary to watch both for visible muscular contraction and for displacement of the umbilicus or middle line. The epigastric, upper, middle, and lower abdominal reflexes should be successively tested. The skin is lightly and rapidly stroked, and care is taken to observe the response to the first stimulus, as in many cases the reflex rapidly disappears on repetition. As the cause of this disappearance does not seem to have been investigated, I recently carried out some observations with the help of Dr. W. Johnson and Dr. E. J. Wood in order to determine the effect of varying physical conditions on the abdominal reflex in normal men.

The first problem to decide was whether the diminution in strength of the reflex on repetition is, as is generally

supposed, the result of fatigue, or whether it is due to some accidental coincident condition, such as the cooling of the skin which must follow exposure to the air. Cooling of the skin proved to be the more important factor. In a man who showed an unusually brisk reflex when his abdomen was first exposed no reflex could be obtained after an interval of five minutes, the surface temperature of the skin having fallen from 33·6° C. to 31° C. in the interval. In a second case the skin on the left side of the abdomen alone was exposed: measuring the reflex on a numerical basis from 0 to a maximum response of 6, it was found, when tested at intervals of a minute, to fall gradually from 3 to 1 at the end of the eighth minute, the surface temperature having fallen from 33·5° C. to 28° C. On now exposing the whole abdomen, the reflex on the right side was found to be still 3 and the surface temperature 33·5° C.

The effect of the temperature of the skin was confirmed by comparing the reflex obtained on the two sides after one had been artificially warmed or cooled. The warmer side always had the brisker reflex. When the temperature of the two sides became gradually more equal as a result of exposure, the reflexes also became more nearly equal. Some inequality was still observed in one case with a difference of surface temperature as small as 1° C., though when it had fallen to 0·4° C. the reflexes were equal; but in another no definite difference could be seen after the temperature of the skin on the side which had been cooled had risen to within 2·5° C. of the other.

It is clear from these experiments that no accurate observations as to the effect of fatigue can be made until the temperature of the skin has fallen to a constant level, as otherwise it would be impossible to separate the effect of cooling from that of fatigue. When the temperature is constant we find that fatigue is only of importance if the skin is stimulated along exactly the same line on each occasion. If care is taken to stimulate different lines, there is no fatigue effect, however near the lines may be. But in the former case the effect of fatigue is very obvious. In one experiment the temperature and reflex having

fallen as a result of exposure from 35° C. to 30° C. and from 6 to 4 respectively, and then remained perfectly constant for a quarter of an hour, the reflex completely disappeared at the thirty-seventh stroke repeated along a line 2½ inches long in a period of thirty-five seconds. The reflex was, however, still as brisk as ever when tested on a line of equal length half an inch away on each side. It is clear, therefore, that the fatigue takes place in the peripheral sense organs, and not in the reflex centre in the spinal cord. Although the effort of fall of temperature is always well marked, that due to fatigue may be very slight or even absent. Thus in one experiment no diminution in the response occurred after thirty-five repetitions along exactly the same line in the course of a minute.

The condition of the bladder was found to exert an unexpected influence on the reflex. In a boy, whose bladder was filled to an extent that made him desire to micturate, no abdominal reflex could be obtained, but directly he had passed eight ounces of urine a brisk reflex was obtained.

In rare instances we observed variations in the reflex which we could not explain ; thus a previously brisk reflex was occasionally found to have almost or completely disappeared after an interval of an hour or two, though there had been no exposure and the bladder was empty.

Inequality of the reflexes on the two sides of the abdomen is often regarded as evidence of organic disease. But before any conclusion can be drawn, it is clear that care must be taken to ascertain whether the surface temperature is approximately equal on the two sides. A patient who had been lying on one side with the lower side in contact with a hot bottle, or one in whom the bed-clothes had been carelessly raised during the examination so as at first to expose one side only, would certainly have unequal abdominal reflexes, which might easily be regarded as of pathological significance, though really due to nothing more than the unequal temperature of the skin on the two sides.

Strümpell and Oppenheim regard the loss of the reflex as a valuable sign in early disseminated sclerosis ; its almost constant presence in normal individuals makes its absence

in doubtful cases distinctly important, but only as a confirmatory sign, as it is very rarely lost before other more conclusive evidence of lateral column disease, especially an extensor plantar reflex, is present. In a series of fifty cases of disseminated sclerosis examined with Dr. W. Johnson at Guy's Hospital in 1912 [1] we found that it was absent in 80 per cent., the plantar reflex being extensor in 82 per cent. on both sides and in 4 per cent. additional cases on one side. In future it will be advisable to warm the skin over the abdomen with a hot bottle, and to see that the bladder is empty before concluding that the abdominal reflexes are really abolished.

As in rare cases it is impossible to obtain one or both of the abdominal reflexes, although the plantar reflexes are normal, it is very important to know whether the reflexes can be lost or become unequal in hysteria. Babinski has laid it down as a definite law that hysteria never leads to any alteration in the cutaneous or deep reflexes, and the majority of authors agree with him. Thus Williamson has recently emphasised the supposed fact that the reflexes are never affected in hysteria as an aid in diagnosing hysterical from organic anæsthesia. A few writers have, however, expressed doubt on the question, and the following clinical and experimental observations prove conclusively that under certain conditions the abdominal reflexes may be unequal or lost in hysteria.

I have already described the case of Private M., the patient who had for nearly two years such complete anæsthesia and analgesia of his skin that no stimulus of any kind, including the strongest faradic current, produced the slightest sensation. The tone of the abdominal muscles was good, the patient was thin, and examinations were made repeatedly over a period of many months under varying conditions, but no abdominal reflex was ever obtained. When at last the patient began to use his left hand, an attempt was made to restore sensation on the left side of his body by the application of a very strong faradic current with a wire brush. After several futile attempts the patient could at length just feel the stimulus

on the left side, but the right side was still totally anæsthetic, and no smaller stimulus produced any sensation on the left side. The same day a definite abdominal reflex was for the first time obtained on the left side, but not on the right side. As sensibility gradually improved the left abdominal reflex became brisker. After several weeks had elapsed, the patient being now able to walk, cutaneous sensibility over the whole body very gradually reappeared without any special treatment. The right abdominal reflex was now obtainable, but it was very much weaker than the left, corresponding with the greater degree of anæsthesia.

We have thus in a single case an example of hysterical anæsthesia, associated at one stage with complete bilateral loss of abdominal reflexes and at another with marked inequality of the reflexes of the two sides.

In 1907 [1] I made several observations on anæsthesia purposely induced by suggestion in the waking state on patients suffering from hysterical paralysis. In a few cases I succeeded in producing a very profound hemi-anæsthesia on the paralysed side, and in these cases the abdominal reflex on the anæsthetic side was weaker than on the other side, and occasionally it was lost, although at the beginning of the experiment the reflexes were normal. When as a result of counter-suggestion the anæsthesia disappeared, the reflex reappeared.

In these investigations a change in the reflex was only observed when the anæsthesia was very profound, and corresponding with this, in the only case of hysteria I have seen in which the abdominal reflexes were affected when the patient first came under my observation, the anæsthesia was complete.

The Plantar Reflex.—The plantar reflex is affected by temperature in exactly the same way as the abdominal reflex, but as the feet are more liable to become cold than the skin over the abdomen, the influence of temperature is of still greater importance. It is well known that absence of the plantar reflex is most commonly due to cold, and that a reflex which cannot be elicited at first may be obtained with more or less ease after restoring the circulation by

exercise or the application of warmth. By noting the surface temperature we found that as it steadily fell on exposure the reflex became less brisk, and finally disappeared. A very slight fall of surface temperature may be sufficient to produce a remarkable alteration in reflex activity ; thus a moderately brisk reflex tested at intervals of sixty seconds became gradually weaker and finally disappeared at the end of the fifth minute, the surface temperature having fallen only 2°—from 28° C. to 26° C.

The diminution and loss of the reflex occur *pari passu* with the diminution in cutaneous sensibility, which is produced, as I have already pointed out, by the peripheral anæmia caused by cold. The loss of the reflex is clearly due to the deficiency of the afferent impulses from the skin, which under normal conditions give rise simultaneously to the sensation of touch and to the plantar reflex. Apart from the cold feet from which healthy individuals suffer, the disuse resulting from paralysis, whether organic or hysterical in nature, is accompanied by diminished circulation and consequent cooling of the surface with loss of the plantar reflex.

In the case I have already described of universal hysterical anæsthesia following stupor the plantar reflexes were, like all the other superficial reflexes, absent. On two occasions I exposed the patient's feet whilst he was asleep. Stroking the soles produced a brisk reflex, and he woke up at the same moment, having apparently felt the touch in his sleep. The return of sensibility in sleep was thus accompanied by return of the corresponding reflex. Immediately afterwards it was found that complete anæsthesia had returned, and no reflex could be obtained. When many months later he recovered from his paraplegia, the anæsthesia slowly but spontaneously disappeared, and at the same time the reflexes could once more be elicited.

It is clear, therefore, that profound hysterical anæsthesia of the sole of the foot may be accompanied by loss of the plantar reflex. This is quite independent of any change in the surface temperature, and must therefore be distinguished from the much more common loss of the plantar

reflex caused by the coldness of the feet in hysterical paraplegia. In the absence of profound hysterical anæsthesia the reflex in these cases returns on warming the feet, just as it does in organic spastic paraplegia if it cannot at first be obtained.

The following conclusions may be drawn from these observations :

(1) The cutaneous reflexes are very easily affected by slight changes in temperature, being weakened and finally disappearing with cold, and being exaggerated by warmth.

(2) Fatigue only leads to a diminution or loss of the reflex if the stimulus is applied along exactly the same line.

(3) The rapid disappearance of the reflex when elicited several times in the course of examining a patient is thus due to the fall of temperature of the skin caused by exposure rather than to fatigue.

(4) Absence or inequality of the superficial reflexes may occur in association with hysterical anæsthesia.

(5) In the absence of profound anæsthesia absence or inequality of the superficial reflexes cannot be regarded as a direct result of hysteria.

(6) Such absence or inequality of the abdominal reflexes, when the skin of the abdomen is uniformly warm and the bladder empty, if repeatedly observed, is very suggestive of organic nervous disease.

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CHAPTER V

SENSIBILITY AND REFLEXES OF MUCOUS MEMBRANES

(1) Conjunctival and Corneal Anæsthesia and Reflexes.—In chapter X I shall refer to the historical case of Madame Stapart, who was cured of hysterical blindness of ten years' duration by a supposed miracle in 1728. The contemporary description of her case shows that the blindness, which affected her left eye, was accompanied by complete anæsthesia of the cornea, conjunctiva, and eyelids, with loss of the corneal and conjunctival reflexes, as the finger could be inserted between the lids without producing any sensation or movement. Directly she was cured, sensibility returned, together with the associated reflexes.

A similar anæsthesia was described by Charcot and others in association with hysterical blindness, and more frequently with unilateral narrowing of the field of vision, the whole being part of a more or less complete hemi-anæsthesia. But none of the numerous patients with hysterical blindness whom I observed during the war complained spontaneously of any loss of sensibility of the eyeball or eyelids, and none showed any evidence of the inflammation, which is liable to follow injury of an eye which has become anæsthetic as the result of an organic lesion. In a small number of my cases, however, anæsthesia was discovered on careful examination. I believe that in these cases the anæsthesia was produced by unconscious suggestion on the part of the observer, as when great care was taken to avoid any possibility of this no anæsthesia was found. A patient with one blind eye is likely to think that the eyeball will also be insensitive to touch if his attention is drawn to the subject,

and he will probably include the eyelids in his conception of the eye as a whole, so that they, too, will become anæsthetic.

In every case in which I have observed hysterical anæsthesia of the conjunctiva and cornea, the corresponding reflexes have been absent and have returned with recovery of sensibility, the association being of exactly the same nature as that observed between cutaneous anæsthesia and loss of reflexes.

In the case of universal and absolute cutaneous anæsthesia following stupor described on page 12, there was total loss of sensibility of the cornea and conjunctiva of both sides, although central vision at any rate was not affected. This case afforded a good opportunity of watching over a long period the slow restoration of sensibility and of corneal and conjunctival reflexes occurring *pari passu.*

(2) Pharyngeal Anæsthesia. — Pharyngeal anæsthesia appears to have been first described as a symptom of hysteria in 1872 by Anstie.[1] "Anæsthesia is very often found when looked for," he wrote, "in one situation where its presence is highly characteristic—the back of the pharynx. If a patient, not taking bromide, can, without retching, let you pass the finger well down to the epiglottis, the diagnosis of hysteria is exceedingly probable." The qualification concerning bromides was thought necessary on account of the observation of Huette in 1850 that the pharynx becomes anæsthetic and the pharyngeal and palatal reflexes disappear in patients taking bromide, an observation which has prompted many physicians to give the drug to patients before examining them with the pharyngoscope, since this procedure was first suggested in 1867 by Gasselin. It can, however, be of little or no real value, as Krosz found in 1876 that it required an enormous dose, such as between 125 and 150 grains taken at one time, to abolish the pharyngeal reflexes, 100 grains never being sufficient. My own observations also show that the supposed effect of bromide on the pharynx, which has been copied from book to book, is non-existent, as careful measurement of the pharyngeal excitability in ten cases of epilepsy before receiving

bromide and after taking between 45 and 80 grains a day, often for several weeks, did not reveal the slightest alteration.

The question is not merely of theoretical interest, as during the last few years I have seen many cases diagnosed as hysterical on account of the presence of pharyngeal anæsthesia. One neurotic young woman barely escaped with her life, because the discovery of pharyngeal anæsthesia led her physician to diagnose an attack of abdominal pain as hysterical in origin, although the delayed operation showed she was suffering from acute appendicitis. The presence of pharyngeal anæsthesia has often been mentioned in reports on war neuroses as an indication of their hysterical nature. It is clear, therefore, that the time has come to decide what value can be placed upon this supposed stigma of hysteria.

With the aid of Captain E. A. Peters and Major J. L. M. Symns, I [2] first drew up a scale of pharyngeal sensibility, beginning with 0, which indicates a complete anæsthesia, and 1, which indicates deficient sensibility and absence of all pharyngeal reflexes, and passing up to 7, which indicates such an extreme degree of hyperæsthesia and such violent reflex spasms that it is quite impossible to examine the throat. After a few days' practice we found our standards were so definite that we always indicated the sensibility in a given case, examined independently, either by the same number or occasionally by numbers differing by one, but never by more. It is not easy to describe the varying reactions, but the following gives an approximate idea of the meaning of each number.

0. Anæsthetic and no reflex.
1. Hardly felt and no reflex.
2. Felt easily and very slight reflex.
3. Slight levator reflex.
4. Good levator and slight tensor reflex.
5. Stronger levator and tensor reflex.
6. Very brisk reflex, making examination very difficult.
7. Maximal reflex, making examination quite impossible.

We then systematically recorded the sensibility in a large number of men, some healthy, some suffering from a great variety of surgical and medical conditions, and others from definite hysterical symptoms, the nature of which was always finally confirmed by their cure by means of

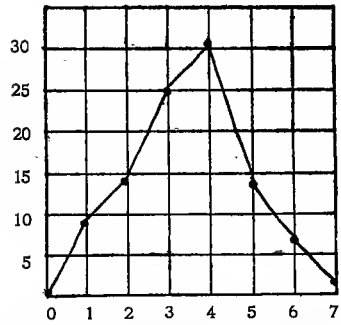

FIG. 16.—Variations of pharyngeal excitability in 170 non-hysterical cases.

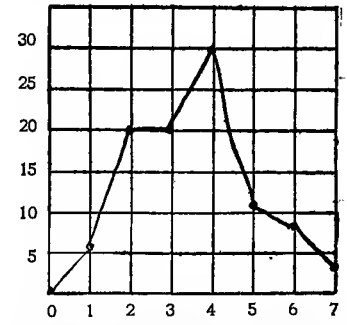

FIG. 17.—Variations in pharyngeal excitability in 64 hysterical cases.

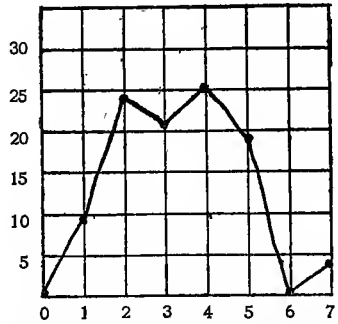

FIG. 18.—Variations in pharyngeal excitability in 24 cases of hysterical aphonia and 10 cases of hysterical mutism.

psychotherapy. We made our examination without indicating to the patient what object we had in view. He was told to open his mouth, and the back of the pharynx and soft palate were then touched with some blunt object, the tongue being depressed when necessary. In the rare cases in which no reflex was produced the man was asked what he had felt during the examination.

The following table shows the pharyngeal sensibility in 170 individuals with no hysterical symptoms and 64 with hysterical symptoms. The cases of hysterical aphonia and mutism, which are included among the latter, are also analysed separately, because it is often asserted that pharyngeal anæsthesia is particularly well marked in hysterical aphonia.

Type of Case.	Degree of Excitability.								Total.
	0	1	2	3	4	5	6	7	
Non-hysterical cases .	1	14	24	42	53	22	12	2	170
Percentage . .	0·7	8	14	25	31	13	7	1·3	100
Hysterical cases . .	0	4	13	13	19	8	5	2	64
Percentage . .	0	6	20	20	30	12	8	3	99
Hysterical aphonia . .	0	3	5	5	5	5	0	1	24
Hysterical mutism . .	0	0	3	2	4	1	0	0	10
Percentage of aphonics and mutes . .	0	9	24	21	25	18	0	3	100

In several cases of hysterical aphonia, as well as of other hysterical symptoms, it was found that the pharyngeal excitability remained unaltered after a cure had been obtained.

These figures and curves show conclusively that pharyngeal sensibility is no more deficient in individuals suffering from hysterical aphonia, or other hysterical symptoms, such as paralysis, contractures, fits, blindness, or deafness, than in individuals who neither are suffering nor have ever suffered in this way, and varies in a similar manner in different people. When care is taken to avoid suggestion, complete pharyngeal anæsthesia is hardly ever found, and the comparatively rare absence of reflexes is met with in normal people just as often as in patients with hysterical symptoms. The single case in which complete pharyngeal anæsthesia was present was that of a stolid individual, convalescent from neurasthenia due to simple exhaustion. He had had no hysterical symptoms, and there was no history of hysterical manifestation in the past. Curiously enough,

in the only example of organic disease in a series of one hundred consecutive cases of aphonia in soldiers,[*] laryngoscopic examination was unusually easy owing to the extremely slight degree of pharyngeal sensibility. If pharyngeal anæsthesia had been taken as a sign of any value, it would thus often have led to erroneous diagnosis.

These investigations were all made on soldiers, but I carried out a similar investigation on a large number of patients at Guy's Hospital a few years ago, and obtained precisely similar results, although about half of the non-hysterical patients and nearly all the hysterical ones were women. Unfortunately the exact figures have been lost, but they did not differ in any way from the present series.

We may thus conclude that pharyngeal anæsthesia is not a stigma of hysteria, and that when it is habitually found by a given observer it must be produced in the majority of the cases by involuntary suggestion on his part. As most patients suffering from hysterical symptoms are abnormally suggestible, it is more likely to be found by careless examination in such individuals than in others. As many individuals suffering from organic disease are equally suggestible, pharyngeal anæsthesia may easily be produced in them. Conversely, in the not uncommon cases seen under the peculiar conditions of modern warfare, in which hysterical symptoms developed as a result of an overwhelmingly powerful suggestion in men who were not abnormally suggestible, pharyngeal anæsthesia would not be produced by careless examination, and the hysterical nature of the symptoms might be doubted if any importance were attributed to pharyngeal anæsthesia as a stigma of hysteria.

In order to explain the frequently repeated assertion that laryngoscopic examinations are particularly easy to make in patients with hysterical aphonia, it must be remembered that about 25 per cent. of patients suffering from aphonia or other hysterical symptoms have, like a similar proportion of normal individuals, a comparatively insensitive pharynx (1 or 2 in our scale). The insensibility would not be remarked upon in a normal individual; but its

association with hysterical symptoms would at once strike an observer who had been taught to regard pharyngeal anæsthesia as a stigma of hysteria, and the one case confirming this would make more impression on his mind than the three in which the pharynx was more sensitive. Moreover, our observations show how easily pharyngeal anæsthesia is produced by suggestion, and a few words of encouragement by the observer would be enough in many cases to render a previously sensitive pharynx insensitive, although the observer might have no notion that his words would have any such effect. In most individuals the pharyngeal sensibility remains unaltered on repeated examination; but in abnormally suggestible men, whether they are actually suffering from hysterical symptoms or not, we have frequently been able to reduce the sensibility from 3, 4, or 5 to 1 or 2 by direct suggestion.

In his book on *Hysterical Disorders of Warfare*, L. R. Yealland states that "it has been my experience to find in conditions of hysterical mutism quite a marked sensory loss over the posterior wall of the pharynx," only strong faradic shocks being recognised; "weaker currents were appreciated in cases of aphonia, and there was no perceptible change in the stammerers." In striking contrast to this, Liébault[4] found diminished or complete loss of pharyngeal sensibility in fifty-two cases of aphonia, but such a degree of hyperæsthesia in fifteen mutes and eight stammerers that he was unable to examine the larynx of any of them. These contradictory results can only be explained by the effect of unconscious suggestion on the part of the two observers, each of whom examined his patients with the idea already fixed in his mind that he would find increased or diminished sensibility, as the case might be, for our observations, described above, prove that the pharyngeal sensibility shows exactly the same variations in mutism, aphonia, and stammering as in normal individuals.

(3) **Rectal Anæsthesia.**—The only other mucous membrane which requires brief consideration is that of the rectum, as numerous French writers, ever since Gendrin first referred to the condition in 1846, have regarded anæsthesia of the

rectal mucous membrane as a cause of constipation. It is curious that not one of them should have taken the trouble to investigate the sensibility of the rectum in normal individuals. In my Goulstonian Lectures on the Sensibility of the Alimentary Canal[5] I described how I had found that the mucous membrane of the rectum is totally insensitive to tactile, thermal, and painful stimuli, so that whatever importance deficient sensibility of the muscular coat of the rectum may have in the production of dyschezia, anæsthesia of the mucous membrane is the normal condition, and neither a result of hysteria nor a cause of constipation.

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CHAPTER VI

CUTANEOUS HYPERÆSTHESIA

If the view be accepted that the physical basis of attention to sensory stimuli is a diminished resistance at the synapses in the sensory tract involved, and that hysterical anæsthesia, with the corresponding loss of reflexes, is caused by inattention, it is easy to explain the occurrence of hysterical hyperæsthesia. When for any reason an individual's attention is fixed to an abnormal degree on sensations coming from a certain area of skin, the resistance at the synapses in the tract conveying impulses from this area to the brain is diminished to a greater extent than under normal conditions. It may be supposed that whilst ordinary attention results in a considerable degree of approximation of the dendrites of adjacent neurones at each synapsis, the excessive attention in hysterical hyperæsthesia results in still greater approximation owing to the extreme degree of projection of the dendritic terminations. Whereas the abnormally great resistance at the synapses in hysterical anæsthesia results in diminution or loss of the corresponding reflexes, the abnormally small resistance at the synapses in hysterical hyperæsthesia results in their exaggeration.

I do not propose to discuss the causes of hysterical hyperæsthesia at any length. But I should like to draw attention to the frequency with which cutaneous hyperæsthesia results from unconscious hetero-suggestion on the part of the observer in exactly the same way that hysterical anæsthesia so often arises. This is seen most frequently in connection with the areas of cutaneous hyperæsthesia which are supposed to occur in association with visceral disease. In my first enthusiasm for the brilliant investi-

gations on the subject by Head, which I read as a student in 1903, I found that I could confirm his observations in every case which I examined. Being particularly interested in the cutaneous hyperæsthesia associated with digestive disorders, I acquired the habit of mapping out Head's area in every case diagnosed as ulcer, and watching its retraction from day to day as improvement occurred, until it finally disappeared, when it was assumed that the ulcer had healed. My first doubt on the subject arose when I had the opportunity in 1909 of seeing ten patients operated on by Moynihan for duodenal ulcer. I had examined them with great care the previous day, and I had entirely failed to find any cutaneous hyperæsthesia, but the ulcer was none the less demonstrated in nine out of ten of the cases at the operation. From that time I have never been able to find any evidence of cutaneous hyperæsthesia in cases of gastric or duodenal ulcer, although I have repeatedly searched for it. Whereas the majority of my earlier cases had occurred among highly suggestible, chlorotic girls, who had up to that time been regarded by most physicians as particularly subject to gastric ulcer, Moynihan's patients were mostly middle-aged, phlegmatic Yorkshiremen. With greater knowledge of the symptoms of ulcer and of the conditions which simulate it, and improved accessory methods of diagnosis with the aid of the X-rays and chemical examination of the gastric contents and fæces, it has become recognised that chronic ulcers are comparatively rare among chlorotic girls, the very people who were formerly supposed to be most subject to them, and in whom hyperæsthesia would be most easily suggested. In examining for hyperæsthesia it must be remembered that unless the greatest care is taken to avoid the possibility of suggestion, it is very easy to produce hyperæsthesia in the area in which it is expected. When improvement occurs under treatment, both the physician and patient expect the area to become smaller; and so, indeed, it does, but not as a result of the healing of the ulcer, but of the physician's hetero- and the patient's auto-suggestion. I have now many times suggested an area of hyperæsthesia away, replaced it by anæsthesia, or transferred

it to the opposite side after it had been carefully marked out by one of my clinical clerks. I have no doubt that the areas of hyperæsthesia had been suggested, just as I had suggested similar areas before I learnt how to avoid doing so.

I do not mean that Head's areas are always produced by the suggestion of an observer looking for hyperæsthesia in a certain zone, when the patient is unduly open to suggestions of the kind owing to abnormal suggestibility and to the presence of symptoms, such as pain, which draw his attention to the area as the probable seat of disease. But I believe that they occur so rarely in diseases of the œsophagus, stomach, intestine, liver, and pancreas as to be of no diagnostic value. Only in diseases of the kidney, which lead to distension of the pelvis, does true cutaneous hyperæsthesia appear to be of some frequency; but even in these conditions the areas of skin involved are much less constant than is often supposed.

It is interesting to note that when areas of cutaneous hyperæsthesia unconsciously suggested in the way I have described are unilateral, the abdominal reflex obtained from the suggested area is brisker than that on the other side, confirming the view I have advanced of the physical basis of hysterical hyperæsthesia. The reflex becomes normal once more when the hyperæsthetic area disappears as a result of suggestion.

CHAPTER VII

HYSTERICAL PAIN

It is a matter of common experience that pain is increased by attention and diminished by inattention. In all probability individual variations in sensibility to pain depend largely upon the varying ease with which the attention can be diverted by an effort of will from the seat of pain. The anatomical basis I have suggested as an explanation of variations in cutaneous sensibility to tactile stimuli can probably be extended to explain variations in sensibility to pain. In this connection I have already referred to the hysterical analgesia which is often associated with hysterical anæsthesia. In the converse condition an individual may be unusually sensitive to pain owing to his inherited mental characteristics, or he may become so owing to exhaustion, insomnia, and sepsis, and the demoralisation which prolonged and excessive pain may induce. This acquired sensibility to pain was comparatively common in soldiers. A man may train himself to such an extent to feel pain that the diminished resistance at the synapses in the tract conveying painful impulses to the brain may persist after the primary cause of pain has been removed. The persistent pain is hysterical, being suggested by the original organic pain, and being curable by psychotherapy.

A patient may continue to feel pain after his diseased appendix has been removed as the result of very slight painful impulses produced in the deep tissues and scar, which an average individual would ignore. The following is one of several cases of the kind which I have observed both in soldiers and civilians. A fuller description of several of these has been published by S. H. Williamson. [1]

Hysterical pain following appendicectomy, cured by psychotherapy.—Private G., aged 24, had had over two years' active service in France and Italy, when he developed an acute attack of appendicitis on July 6, 1918. He was operated upon the same night, two tubes being inserted. He was transferred to a hospital in England, and was allowed to get up on August 16. Since then he had had constant sharp pain in the region of the scar. He was sent to his depot, and told that the pain would go in time. He was given clerical work, but could not do it, and was transferred to Seale Hayne Hospital, under Captain S. H. Wilkinson, on December 21, 1918. He complained of constant pain in the region of the scar, especially when in bed. In a long therapeutic conversation it was explained to him that the original pain had made a great impression on his mind, because at the time he was physically and mentally tired from constant active service for over two years; in spite of the fact that the cause of the pain had gone, his mind had got so accustomed to feeling the pain that he continued to feel it. The pain disappeared completely, and two days later he was given work in the farm. There had been no return of pain when he was discharged a month later.

The pain felt in the scars of wounds long after they have healed is, I believe, in many instances of the same nature. It has generally been ascribed to nerve fibres becoming involved in scar tissue, but nothing abnormal may be found at operation, or complete excision of the scar may produce no improvement, the pain persisting in the new scar. We saw numerous cases of this kind, which were often associated with hysterical paralysis or contracture, in which severe pain was rapidly cured by psychotherapy.

Hysterical painful scar following a compound fracture, cured by psychotherapy.—Driver L., aged 19, was kicked by a horse on the left leg on August 6, 1918, and sustained a compound fracture of the tibia and fibula. He was in bed for eight weeks, and when allowed up complained of constant pain over the fracture. He was admitted to Seale Hayne Hospital, under Captain S. H. Wilkinson, on

November 12, 1918, walking with a marked limp, tilting his pelvis down to the left with every step he took, and complaining of severe pain over the scar, which was red and tender. It was explained to him that as soon as his gait was corrected and he walked properly he would find that his pain would disappear. In twenty minutes he was running quite normally, and admitted that he had no pain. When discharged he was fit in every way, having had no return of pain during the month he had been working on the farm.

It is generally assumed that the headache following the explosion of a shell in the immediate neighbourhood, especially if consciousness was lost, is due to concussion. If this is correct, so-called "shell-shock headache" is of exactly the same nature as the headache which follows concussion in civil life. It is well known that the most effective treatment of the latter is rest, but that if insufficient rest is taken in the earliest stage the headache is likely to be prolonged for an indefinite period. This has proved to be the case in "shell-shock headache," and all who have had such cases under their care must have been struck by the extreme difficulty of relieving the headache by ordinary means. The analgesic drugs, which give, at any rate, temporary relief for most forms of headache, are either entirely without effect or only produce a slight amelioration of the pain. If a considerable period has elapsed since the onset of the headache, during which the patient has been getting about, a renewed period of rest is generally of comparatively little use. In a number of cases I was struck by the complete relief which immediately followed a lumbar puncture, but unfortunately this was rarely permanent, and often only lasted for a day or two. I was at the time unable to explain this, as the fluid was never under increased pressure, so that it did not seem possible that the improvement was due to the reduction of an abnormally high intracranial pressure.

The following case seems to give a clue to the explanation of the persistence of the headache in some of these cases. [1]

"*Shell-shock*" *headache, cured by hypnotism.*—Corporal

H. served from August, 1914, and remained well until he was blown up in November, 1917. He was away from his unit for a month on account of a stammer and tremor. He then returned to duty, but was wounded in April, 1918. He was now sent to hospital, and after six months' treatment for neurasthenia was admitted to Seale Hayne Hospital, under Captain C. H. Ripman, in October, 1918. He was shaky, and complained of a heavy pain in the head during the day, which had been present ever since he was blown up. During the whole night he sat up in bed holding his head and crying with pain. In spite of the severity of the pain he was not inclined to make much of it during the day. He was treated with drugs, none of which had any effect. Hypnosis was consequently tried. The headaches improved at once. After the first treatment he slept through the night, and he had no headache the next day. After three treatments he felt perfectly well, and became one of the most lively men in the ward, having previously spent all day curled up on his bed and all night crying with pain. He was discharged a month later, and wrote in February 1919 to say that there had been no return of his headache.

In this case, in spite of the extreme severity of the headache, complete relief was produced by suggestion without any accessory factor coming into play. It is inconceivable that suggestion, by which is meant the communication of a proposition without giving adequate and logical grounds for its acceptance, could have produced such a result in a headache of organic origin. Consideration of this case points to the possibility that the relief produced in our earlier cases by lumbar puncture was due to suggestion, and the absence of any relief with ordinary analgesic drugs could be explained on the assumption that the headache was of entirely psychical origin. It is probable, therefore, that the long-continued and intractable headache following the concussion caused by a high-explosive shell is frequently caused by the perpetuation by auto-suggestion of a headache, which is at first organic in origin and a genuine result of concussion. Being produced by suggestion and cured by psychotherapy, it can be correctly called hysterical.

The chronic headache which follows concussion in civil life is probably often of similarly hysterical origin.

Hysterical headache following concussion.—A lady, 49 years old, an artist by profession, fell on her head in February 1917 as the result of a collision with a motor-lorry whilst she was driving a dog-cart. She was badly shaken, but was not rendered unconscious. Severe headache developed immediately afterwards. It persisted all day, and at night prevented her sleeping. She did not improve in spite of wearing spectacles for astigmatism and giving up painting and reading since her accident. In October 1918 she was told that the pain was rheumatic, and was ordered a strict diet and massage for her head. After this she became terrified of cold and damp, and kept indoors almost all the time; but whenever the weather was bad, the headache was much aggravated, although this had not hitherto been the case.

I saw her for the first time on March 1, 1919, when I explained to her that the headache was simply a perpetuation of that due to the original concussion. I told her that as the effects of the latter had long ago passed away she would no longer have any headache if she ceased to expect it. I explained to her that it was clearly not due to her astigmatism, as her glasses had not helped her, and she was no better when she did not read or paint, and that the failure of her dietetic precautions and staying indoors had proved that it was not rheumatic. On my advice she gave up her glasses, and began to read and paint, and she went out whatever the weather was like. I saw her a week later, and she had only had one slight headache, which had come on during a rain-storm, this having been apparently again suggested by the weather. Further explanation resulted in complete disappearance of the headache, in spite of taking no further precautions to prevent it. She was given no drugs, and the various medicines she had taken before, both during the day and at night for her insomnia, were discontinued. From the first conversation she slept perfectly well. She wrote on April 16 to say she was still free from headache.

In the following case the primary pain was caused by gross organic disease.

Hysterical pain following the removal of a painful growth.—A man of 63 had been suffering for some weeks from slight supra-orbital neuralgia on the right side, when he was knocked down by a cyclist and rendered unconscious for thirty-six hours. After this the pain was greatly exaggerated, and produced severe insomnia. Six months later the right antrum was found to contain pus; after it had been drained, it was discovered that the abscess was secondary to a growth, which was subsequently removed completely. Instead of disappearing, the pain became worse than ever. It persisted in spite of two injections of alcohol, which produced anæsthesia in the area supplied by the lower two branches of the trigeminal nerve. Numerous drugs were tried without benefit, and the patient became progressively more demoralised, until four months after the operation he was continually screaming out with pain, and could get no sleep either by day or night. He took very little food, and was in a condition of profound exhaustion when, at this stage, I saw him for the first time. As there was no sign of recurrence of the growth, it appeared possible that the pain was due to the hysterical perpetuation and exaggeration of what was at first organic. The growth had probably caused the original neuralgia, and this was aggravated by the accident which had involved the antrum. As it was quite impossible to reason with the patient, I attempted to hypnotise him. I did this with much less difficulty than I had anticipated, and I then suggested that he would have no more pain, but would have a long sleep. During the next twenty-four hours he kept quite quiet, and slept the greater part of the time. I hypnotised him on four other occasions, and also tried to make him understand whilst he was awake the nature of his pain, and how he could control it. It is four months since I first saw him, and although there have been occasional slight recurrences of pain, these have been insignificant, and he continues to sleep well, and now takes an interest in his surroundings, no further drugs having been required.

Hysterical pain is a very real thing, and cannot be distinguished from pain caused by organic disease by its character, or by the absence of associated sympathetic phenomena. It may, for example, give rise to tachycardia, dilatation of the pupils, pallor, and sweating, if the original organic pain did so, because the afferent tract of the reflex paths involved offers so little resistance to impulses excited by peripheral stimuli that the latter still produce marked effects, even when they are so slight that if the patient would cease to expect pain and thus allow the normal resistance to return, they would either produce no effect at all or only an insignificant amount of pain and little or no sympathetic reflexes.

REFERENCES

[1] S. H. Wilkinson, *Seale Hayne Neurological Studies*, i. 327, 1920.

[2] A. F. Hurst and C. H. Ripman, *Seale Hayne Neurological Studies*, i. 193, 1919.

CHAPTER VIII

HEARING, LISTENING, AND HYSTERICAL DEAFNESS

HYSTERICAL deafness is probably less rare in civil life than has generally been supposed. In this chapter its etiology will be primarily discussed in the light of experience gained from the observation of numerous cases occurring in soldiers, among whom it was comparatively common, and of a small number of severe cases in civilians.

The noise and concussion produced by the explosion of a shell of high power in the near neighbourhood frequently caused deafness. The patient was dazed or unconscious as a result of the explosion, and when his mind became clear again he discovered that he could not hear. Both ears were generally affected, but the one on the side more exposed to the explosion of the shell was often deafer than the other. The initial deafness was doubtless due to concussion of the internal ear, as it was sometimes associated with vertigo and temporary nystagmus. One or both drums were often perforated owing to the sudden enormous change in atmospheric pressure. Such a perforation did not greatly affect the prognosis, as the tear generally healed, and if no hysteria developed normal hearing was restored. Sometimes, perhaps, the ossicles were dislocated by the force of the explosion, in which case some permanent impairment of hearing would result.

The deafness might pass off in the course of a few hours, but more frequently it lasted for a few days. If it persisted for a still longer period, it was almost always hysterical, at any rate in part. The initial concussion deafness made such an impression on the mind of the soldier that, on

coming to himself, whether he had actually lost consciousness or not, his first thought was for his hearing, and he might be so convinced that he was permanently deafened that he became actually deaf as a result of auto-suggestion. This was especially likely to be the case if for any reason the idea of deafness had previously entered his mind; it was for this reason that a large proportion of cases occurred in men who had old disease of the ear.

The temporary deafness, which was a familiar condition before the war, both in gunners and other people who were exposed to the frequent repetition of loud noises, might in the same way become perpetuated and exaggerated by auto-suggestion.

Lastly, organic deafness, especially if the onset is acute, as in that due to involvement of the auditory nerve trunk in cerebro-spinal meningitis, may remain complete after the disappearance of the active disease has been followed by sufficient restoration of the damaged structures for a certain amount of hearing to have returned. This, again, is due to auto-suggestion, the final deafness being organic with a superadded hysterical element, which is capable of removal, like all hysterical symptoms, by psychotherapy.

If left untreated without any encouragement, and especially if steps are taken to teach the patient lip-reading, he will become more and more convinced that he is permanently deaf, the effect of the original auto-suggestion being increased by the unconscious hetero-suggestion of those in charge of the patient. One of the worst cases I have seen was that of a man, who was told by an aurist that his case was hopeless because he had already been deaf for four months without any improvement occurring. Prolonged psychotherapy was required to cure him. If he had been told that the kind of deafness from which he suffered never lasted for more than four months, and that he would certainly be well in a week, rapid recovery would have resulted.

Pathogenesis.—Hearing does not consist merely of the perception of impulses conveyed to the brain when the ear

is stimulated by sound waves. It is an active process, and in order that sounds may be heard the individual must listen. Inattention during a dull sermon results not only in absence of perception of the words said, but of total deafness to the sound of the preacher's voice. It is clear that impulses to the cortical centre of hearing must be actually interrupted by inattention. The most satisfactory theory is that in the act of listening the resistance at the various synapses in the auditory path becomes diminished by some such process as a throwing out of dendrites, which brings those of contiguous neurones into more intimate connection. In inattention the synapses are unswitched, the resistance being increased by the retraction of the dendrites.

In animals there is an obvious motor element in listening in addition to the sensory element just considered. This is seen in the movements of the external ear, which is most obvious in those which depend upon flight, prompted by the acuity of their hearing, for safety from their enemies. If one pays attention to one's own sensations in the act of listening, it appears as if some active motor process was taking place in the ear. This probably consists in the simultaneous contraction of the stapedius and tensor tympani muscles, which exert tension on the chain of ossicles in opposite directions. Their function appears to be to regulate the balance of the stapes, and so modify the intravestibular pressure that the cochlea may act at its maximal efficiency. The small muscles of the external ear are generally regarded as rudimentary and functionless structures in man. Keith has pointed out that muscles which are never used tend in the course of many generations to disappear. But the muscles of the external ear are invariably found in man, and therefore differ from those rudimentary muscles which have either disappeared altogether or are only found in a minority of human beings. They are, moreover, as well developed in man as in apes. It thus appears probable that they are not so inactive as is generally supposed, and that their activity is not confined to the occasional display of their power by the comparatively

small number of individuals who can voluntarily move their ears.

How strong the muscles may be was well shown in the case of a soldier, who constantly saw in his dreams a German whom he had bayoneted in the face; at the same time he developed a bilateral spasm of his face, which recurred every few seconds. Every muscle supplied by the facial nerve was involved, including those of the ear, the movements of which were extraordinarily well marked, although the patient had never been able to move his ears voluntarily. When the dreams disappeared as a result of psychotherapy, the spasms also ceased.

On closely watching the ears of a man who was listening intently, I found that a definite movement always took place. I have since confirmed this in several individuals. In some cases the outer edge of the auricle moved outwards and forwards on listening to a sound in front, and inwards towards the side of the head on listening to a sound behind. The most vigorous movements I have seen occurred in a man who could move his ear voluntarily, and who was also aware that he moved it when he listened intently. It is, of course, obvious that these movements are mere remnants of the big movements in animals, which have the object of concentrating as many sound waves as possible in the external auditory meatus, but they are sufficient to explain the constant presence of the muscles in human beings.

The contraction of the stapedius and tensor tympani might be thought to play a more important part, but careful observations made with W. M. Mollison failed to show the slightest alteration in the process of hearing in complete unilateral facial paralysis, including presumably the stapedius muscle.

When a man is temporarily deafened by a loud noise or by some recoverable disease, he finds that he is unable to hear, however much he tries, and consequently after a time he gives up trying. That is to say he ceases to listen, and when the cause of the deafness at last disappears, he has become so convinced that he cannot hear that he makes no further attempt to listen. Although the sound vibrations

reach his ears in the normal way, they do not give rise to the slightest auditory sensation because of this inattention : he is then suffering from hysterical deafness, the inability to hear having been suggested by the original organic, though temporary, deafness. Severe hysterical deafness developed in a soldier while he was the only Englishman in a German prison ; he ceased to pay attention to what was said, as he could understand nothing, and in time he ceased even to hear the unintelligible conversation of his companions. His hysterical deafness was rapidly cured by psychotherapy when a year later he came under the care of Major J. F. Venables at the Seale Hayne Hospital.

In hysterical deafness the synapses at one or more of the cell-stations in the auditory path to the cerebral cortex (fig. 19) must therefore be unswitched, possibly as a result of retraction of the dendrites. Further evidence for this follows from a study of the auditory-motor reflex.

Auditory-motor or Jump Reflex.—A sudden noise normally causes an individual to jump, and often to blink, and the pupils dilate ; the " jump," at any rate, is a protective reflex, and represents the preparation for flight or fight. The blinking cannot be suppressed by the majority of people, although expert revolver-shots are said to acquire inhibitory power over it. It is very doubtful, however, whether they ever suppress the pupil reaction.

An officer, whose left motor cortex had been almost completely destroyed, went to the *Man that Stayed at Home* about four months after he was wounded. His right arm jumped violently when the gunshot rang out on the stage, although no trace of voluntary movement returned until three months later. The efferent part of the reflex is thus sub-cortical. In certain war neuroses of emotional origin, in which the reflex is exaggerated, jumping continues during sleep and deep hypnosis, although the patient does not hear the noise which induces it even in a dream. The afferent part of the reflex is thus also sub-cortical, the reflex being quite independent of actual hearing.

Experiments on animals by Sherrington and Forbes[1] confirm the conclusion we reached from clinical observations

—that the auditory-motor reflex is a function of the mid-brain. They showed that both the posterior corpus quadrigeminum and the medial corpus geniculatum are concerned.

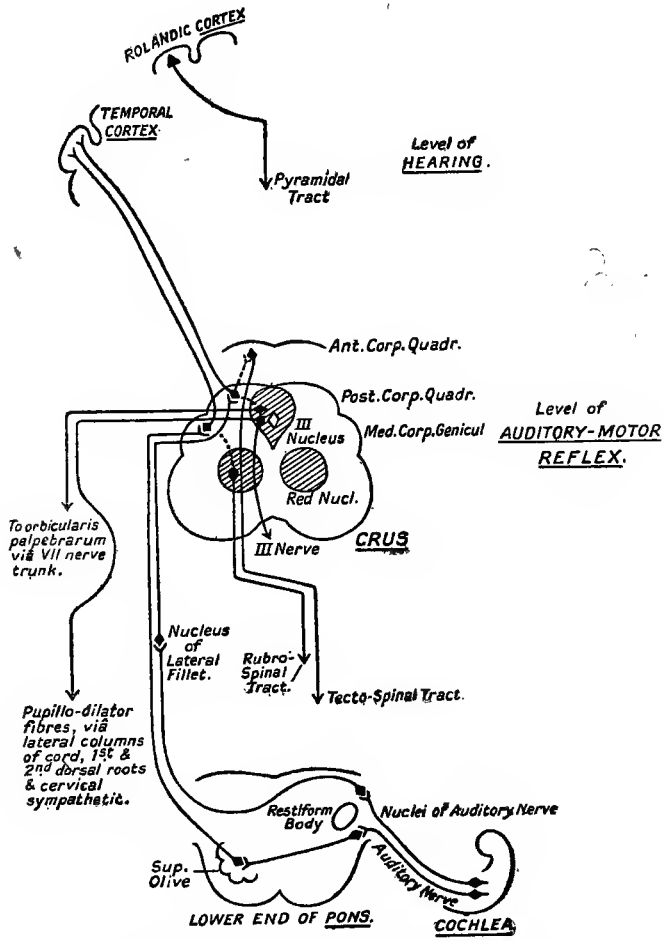

FIG. 19.—Diagram illustrating neurones concerned in hysterical deafness and in the auditory-motor reflex.

The close relation of these centres to the blinking and the sympathetic pupilo-dilator centres in the neighbourhood of the third nerve nucleus, and to the anterior corpus quadrigeminum and red nucleus, in each of which a de-

scending motor tract originates, gives an anatomical basis for this view (fig. 19). In the experiments of Sherrington and Forbes on cats, sounds, especially barks, yowls, and whistles of birds, excited orientatic reflex movements of the pinna and neck after the complete removal of the cerebral hemispheres, striata, and thalamus. Reflex movements expressive of anger and aggression—lashing of the tail with bristling of its hairs, and flexion and extension movements of the limbs—were also produced. In our cases of complete bilateral deafness the auditory-motor reflex was completely absent, but returned simultaneously with the restoration of hearing as a result of psychotherapy. A very nervous but totally deaf mute remained completely unmoved, never jumping or showing a flicker of his eyelids during one of the severest thunderstorms I have known, and yet the next day he was completely cured by suggestion under partial anæsthesia.

A slight reflex was present in most cases of severe but incomplete hysterical deafness, but it became less marked and sometimes disappeared completely or was confined to a slight dilation of the pupil when the test was repeated.

The abolition of the auditory-motor reflex in absolute hysterical deafness makes it clear that one or more of the unswitched synapses in hysterical deafness must be at the level of the reflex, or still lower—in the auditory nucleus or one of the intermediate cell-stations, the superior olive and the nucleus of the lateral fillet, or perhaps in all (fig. 19).

The persistence of the deafness during hypnosis and natural sleep shows that when the inattention of hysterical deafness has lasted for a considerable period, the unswitching of the synapses is more profound than that which normally occurs during deep sleep, in which the synapses can always be forced by a loud noise.

Diagnosis.—In the observations I carried out at Netley with E. A. Peters [8] we found it necessary to discard almost all the criteria formerly used in the diagnosis of organic deafness from deafness due to hysteria or malingering.

(1) *History.*—Complete bilateral deafness following the

explosion of a powerful shell was generally hysterical, though a lesser degree of asymmetrical organic deafness of a more or less permanent nature might be produced at the same time owing to perforation of the drum or hæmorrhage into the middle ear, both of which were often followed by otitis media, or to dislocation of the ossicles. It is still doubtful whether absolute organic deafness ever results from hæmorrhage into the internal ear following aerial concussion, as no anatomical evidence of such an occurrence has yet been forthcoming. Deafness following an acute illness, especially cerebro-spinal meningitis, is at any rate in part always organic, but we have found that the deafness may be increased as a result of auto-suggestion, no spontaneous improvement in function occurring when the anatomical condition improves.

(2) *Other evidence of hysteria.*—Our investigations have shown that hysterical symptoms may develop in the most normal individuals if the suggestion which provokes the symptoms is sufficiently powerful. No more powerful suggestion of deafness could be imagined than the organic but temporary deafness caused by the noise of an explosion in the immediate neighbourhood. It is therefore not surprising that hysterical deafness has occurred almost as frequently in soldiers who have no personal or family history of neuroses as in those with a neuropathic predisposition. Deafness has consequently been the only hysterical symptom in many of the cases. When, however, the onset was associated with extremely terrifying circumstances, some of the physical results of fear, such as mutism and tremor, were often perpetuated as hysterical symptoms. Thus hysterical deaf mutism was quite common. In two cases sand was thrown into the patient's eyes from the sand-bags on which the shell exploded, and the hysterical deafness was accompanied by hysterical blindness, which followed the conjunctivitis caused by the irritation of the eyes with the sand.

The only conclusion which can be drawn from these facts is that while the association of deafness with mutism or with tremor is very suggestive of hysteria, hysterical deafness

occurs most frequently in otherwise normal individuals with no symptoms or history of neuroses.

(3) *The supposed association of hysterical anæsthesia of the external ear with hysterical deafness.*—Ever since Briquet[3] in 1859 described the association of hysterical deafness with anæsthesia of the external ear, this has been regarded as an almost constant phenomenon. The accepted teaching of neurologists on the question may be gathered from the article on hysteria by Oppenheim in his *Lehrbuch der Nervenkrankheiten*, in which he states that hysterical deafness is frequently accompanied by localised anæsthesia of the external ear, unless anæsthesia of the whole side of the body is present. As recently as March 1918, Yealland, in his book on *Hysterical Disorders of the War*, states that in hysterical deafness "as a rule there is some sensory loss to pin-pricks over the skin covering the mastoids." Among aurists Politzer refers to the same association of symptoms.

J. L. M. Symns, R. Gainsborough, and I [4] noted the sensibility of the external ear in a series of twenty-nine soldiers, all suffering from organic deafness, due to various causes, and of varying degrees of severity. The ears were first touched lightly with a finger or some wool, sometimes beginning with the deafer ear, sometimes with the ear which was less deaf or normal; at the same time the patient was asked, "Which side do you feel more distinctly?" A similar comparison was then made between each ear and the cheek on the same side. In twenty-eight out of the twenty-nine cases hearing was distinctly better on one side than the other, and in several the latter was normal. Eighteen of the twenty-eight had partial anæsthesia, and four had hyperæsthesia of the affected ear; the better ear was normal in every case. In the single case in which the deafness was equal in the two ears there was no anæsthesia; but this man did not appreciate that he was deaf at all, a slight degree of deafness having only been discovered when the ears were examined on account of other symptoms. The six cases of asymmetrical deafness, in which no anæsthesia or hyperæsthesia was found at the first examination, were then examined again, but a gross form of suggestion

was now employed, the patient being asked the following questions : " You feel my finger less clearly when I touch this ear (the deafer one) than this one, do you not ?" and " You feel me touch this ear (the deafer one) less clearly than your cheek, do you not ?" Four out of the six patients answered " Yes " ; in the remaining two the degree of deafness was very slight. In several of the patients anæsthesia was found to be very well marked, and was associated with complete or partial analgesia. So real was this loss of sensation that one patient was subsequently seen putting a pin through the lobe of his ear for the amusement of the other men in his ward.

In a case of severe bilateral hysterical deafness examined in the same way, both ears were found to be completely anæsthetic. In a second case, in which one ear was totally deaf owing to organic disease and the other had been totally deaf owing to hysteria, but was now only partially deaf as a result of psychotherapy, the organically deaf ear was found to be anæsthetic, and the hysterically deaf ear was normal, corresponding with the fact that the deafness of the former was much more severe than that of the latter. A gun-layer with severe hysterical deafness on an organic basis, but with no other nervous symptoms, who was totally deaf in one ear and almost so in the other, showed no difference between the sensibility of the external ear on the two sides, both being perfectly normal, and attempts to suggest a deficiency completely failed.

It is thus clear that the supposed association of hysterical anæsthesia of the external ear with hysterical deafness is a complete fallacy, and that anæsthesia is likely to occur in a deaf ear if looked for, whether the deafness is organic or hysterical, so long as the individual is sufficiently suggestible and not too well educated. Thus in all the above cases the patients were soldiers who had been on active service, and our experience has shown how remarkably suggestible the majority of war-worn soldiers are, even in the absence of any hysterical or other nervous symptoms. The results obtained with ordinary hospital patients was consequently less striking. Among four adult male civilians,

three females, and two boys, all of whom were deaf in one ear or deafer in one than in the other, only one of the adult males had an anæsthetic external ear. When, however, they were shown two test-tubes, one of which was said to contain hotter water than the other, and were asked to say which side was touched with the hotter tube, eight out of nine said that the deaf (or deafer) ear felt the heat more than the other ear, although in every case the same tube was used for touching both ears.

Ten well-educated individuals, who were asked whether they would expect any difference between the sensibility to touch in the two external ears if they were deaf on one side only, replied in the negative. On the other hand, seven indifferently educated men all replied in the affirmative. The difference is simply due to the fact that no intelligent man would expect his external ear to be anæsthetic if he were deaf, but a man of less education would act upon the suggestion implied in the question without criticising it. The greater frequency of grotesque hysterical symptoms among hospital than private patients, and among private soldiers than officers, is doubtless due to the greater intelligence and greater development of the critical faculties to the latter.

(4) *Bone and air conduction.*—When deafness is not absolute, a tuning-fork can still be heard by bone conduction (positive Rinne's test). This shows that the deafness does not depend on changes in the middle ear, even when these are present. But it does not distinguish between the nerve deafness due to organic disease and that due to hysteria. Moreover, the test can only be applied in the slighter cases, as in many instances deafness is absolute and no hearing is possible, whether the sound is conducted by bone or through the air. The vibration of a tuning-fork held on the mastoid process is, however, often felt, and the aerial vibration caused by very loud noises is occasionally appreciated, even when no sound is heard.

(5) *Auditory-motor reflex.*—In absolute deafness, whether due to organic disease or hysteria, the auditory-motor reflex is absent, but in partial deafness, whether organic or

hysterical, if a sound can be heard at all, it may produce a reflex. If a reflex is present, but the patient says he can hear nothing at all, he is probably a malingerer, but so long as he admits that he can hear something, the test does not help in distinguishing between organic deafness, hysterical deafness, and malingering.

(6) *Persistence during sleep.*—As hysterical symptoms are due to suggestion, it might be expected that they would not persist during sleep, and Babinski regards this as a definite law. My experience agrees with his with regard to all other hysterical symptoms which I have investigated, such as paralysis, contractures, and anæsthesia. I have seen several deaf mutes, and one aphonic, who talked naturally in their sleep, and a man with hysterical amnesia had nightmares referring to his period of amnesia. But hysterical deafness, the behaviour of which during sleep does not appear to have been tested by Babinski, is an exception. Thus, greatly to my surprise, I found it quite impossible to wake two of my patients, who were suffering from total hysterical deafness, and were sleeping in a hut by themselves, by shouting or by making other very loud noises within a foot of their heads. I convinced myself that deception was impossible, and the hysterical nature of the deafness in both cases was at a later date conclusively proved by their instantaneous recovery with powerful suggestion. In one patient a slight twitch of the eyelids was sometimes observed with a particularly loud noise, but not in the other. It seems probable that a malingerer could be detected by this test, as he would certainly wake if a loud noise was made when he was asleep, whereas in hysterical and organic deafness waking does not follow.

(7) *Effect of hypnosis.*—I had expected that hearing would return in hysterical cases during hypnosis, but I found it quite impossible to make deaf patients, whom I had deeply hypnotised, obey any command or show any signs of hearing, and no auditory-motor reflex was produced. The unswitched synapses thus appear to remain unswitched during hypnosis, as they do during sleep.

(8) *Character of the voice.*—In almost all cases of severe deafness due to organic disease the character of the voice changes. It is difficult to understand why there should be any difference in the effect of total deafness on the voice, whether it is organic or hysterical, as the change is simply a result of the patient's inability to hear his own voice. Although in some of our hysterical cases the typical voice of the organically deaf developed, the majority showed no change in timbre or intonation. In the three cases I have seen which dated from early infancy, the patients had learnt to talk, but their speech was of the very indistinct type which is characteristic of deaf-mutes who have been taught to speak without ever having heard.

(9) *Lip-reading.*—When a deaf man teaches himself lip-reading, his deafness must be of a high degree. Several patients with hysterical deafness learnt it with remarkable rapidity, so that it cannot be regarded, as has been suggested, as a sign of organic disease. On the other hand, it is very unlikely that a malingerer would ever learn lip-reading.

(10) *Vestibular symptoms and reactions.*—Disturbances in the vestibule as a result of concussion may cause spontaneous nystagmus, which may be accompanied by giddiness and staggering, but these symptoms rarely last for more than a few hours.

We found that the only test upon which almost complete reliance can be placed in the diagnosis of absolute hysterical deafness from absolute organic deafness is the presence of normal vestibular reactions in the former and their loss in the latter. The vestibular reactions are entirely beyond the control of the will, and it is therefore inconceivable that they should disappear as a result of suggestion. As hysterical symptoms are always caused by suggestion, the vestibular reactions must remain unaffected in hysterical deafness. On the other hand, it is highly improbable that any organic lesion could damage the cochlea or the cochlear nerve or nucleus on both sides sufficiently to cause total bilateral deafness without at the same time damaging the vestibules or vestibular nerves or nuclei, so that total

organic deafness is almost certain to be accompanied by deficiency in the vestibular reactions. The only exception of which I have heard is a case reported by Fraser [5] of a congenital deaf man, in which microscopical examination of both inner ears showed that the organ of Corti was so malformed that hearing was almost abolished, while the vestibules were normal. The caloric reaction was noted on both sides some days before death, and a normal response was obtained.

The vestibular test has proved of great value in cases of total deafness. But it must be remembered that in partial organic deafness the vestibular reactions may be lost in severer cases and exaggerated in slighter cases; any hysterical element which may be present in such cases can only be recognised by the improvement which follows psychotherapy.

The vestibular reactions may be investigated by the rotation, caloric, or electrical tests, in all of which nystagmus and giddiness occur in normal individuals, but not if the vestibules or vestibular nerves are damaged. We have generally employed the rotation test, as it requires no special apparatus, the patient turning rapidly round five times in one direction and subsequently in the opposite direction. The character and degree of the nystagmus on looking in the direction opposite to the rotation is estimated, and the subjective and objective evidence of vertigo is investigated. The rapidity of the movements of the eyes and their duration should be the same when the individual is rotated clockwise or counter-clockwise. If any inequality is observed, one vestibule must be involved and the other spared, or one must be involved more than the other. An equally simple and satisfactory test [6] is for the patient to turn five times round a walking-stick, which he holds in the erect position, with his back bent so that his forehead rests upon the handle of the stick; he then attempts to walk along a straight line marked on the floor. A normal individual diverges from the line in the same direction as he has been turning, the angle being equal whether he turns to the right or left; but in severe bilateral vestibular disease he walks straight

forward, or if the ears are unequally affected, the angle differs according to the direction he turns.

The following remarkable case well illustrates the fallacious character of the criteria which were formerly regarded as sufficient to make a definite diagnosis of organic deafness.

Absolute functional deafness in a young man of eighteen dating from infancy.—G. C., a deaf-mute, 18 years old, appears to have become deaf after a fall on his head some time between the age of three and nine months. His mother is certain that he could hear as a small baby, but she noticed that he was deaf before he was a year old. Sir Dundas Grant saw him when he was three years old. He has kindly looked up his old notes, from which it appears that although there was then "a little trace of hearing," he concluded that he must "be classed with the deaf-mutes." With great difficulty he was taught to speak, but he has the characteristic, extremely indistinct speech of a deaf-mute. He does not remember ever having heard anything at all, except on rare occasions during the last few years, when he thinks he has sometimes heard a loud noise, though possibly he really only felt the vibrations of the sound. When I first saw him in May 1919, he appeared to be totally deaf, but there were no signs of middle ear disease, and the vestibular reactions were perfectly normal. The auditory-motor reflex was completely absent. I explained to him by means of lip-reading, at which he is an expert, that he could not hear because he had never tried to listen, and if he once made an effort to listen he would begin to hear. On this first visit he was taught to listen sufficiently to hear his name called close to his ear; this was, so far as he remembered, the first word he had ever heard. The same day he heard his bicycle bell and a motor horn for the first time. During the course of the next three weeks, in which it was impossible to give him more than an occasional short lesson, he learned to hear a number of words, each of which had to be taught separately, as although he could hear the sound, it conveyed nothing to him until he realised what the word was by lip-reading. When once learnt he could understand it on a future occasion, but found it extremely difficult to continue

to listen for more than a few minutes, so progress was slow. At his best he could hear a familiar word from the other side of the room without the voice being raised, but frequently it was necessary to shout a word into his ear. At the same time he improved sufficiently to be able to hear all ordinary sounds. He could hear a band, and he could hear notes played on the piano, but he had no idea of pitch, and could not distinguish one note from another. The auditory-motor reflex was now normal.

He is still making slow but steady progress. His father says that he now almost always looks up at once when anybody speaks, and that he seems to hear most sounds without any obvious effort. The diminished resistance in his auditory tract is further shown by the fact that he now sometimes wakes when a cock crows, although before treatment was begun the deafness was complete when he was asleep as well as when he was awake. When I last saw him on July 20, 1920, he could hear a watch ticking three inches away from his left ear, and could repeat words he had never heard before if they were spoken several times distinctly, but not loudly, near his ear. At the same time his speech has slightly improved.

Although his hearing is still far from normal, there is every reason to hope that considerable further improvement will take place.

In this case a young man of eighteen had been a deaf-mute from infancy, and the many aurists who had seen him in the past regarded his condition as obviously incurable; the complete absence of anything in himself or his relations which could be regarded as pointing to a tendency to neuroses, the absolute loss of bone and air conduction and of the auditory-motor reflex, the persistence of deafness during sleep, the characteristic speech of the deaf-mute who has painfully acquired the power of making himself understood, and his great skill as a lip-reader, might have been taken to prove beyond the shadow of a doubt that the deafness was organic. But the vestibular reactions were normal, and acting upon this alone, we were able to prove the functional nature of the condition by restoring his

powers of hearing by means of psychotherapy. The deafness which developed when he was about three months old must have been due to some organic but evanescent lesion. It occurred at a period when he was just learning to listen, and consequently interrupted his development in this direction. When the organic cause disappeared he had become unaccustomed to listen, and he never learnt to do so again. The deafness was thus really functional and therefore capable of being cured by psychotherapy.

A proper understanding of the psychological process involved in listening, and of the disturbance in the process which results in hysterical deafness, should lead to a great improvement in the treatment of the latter. When hysterical deafness is associated with mutism it requires no special treatment, as hearing almost invariably returns spontaneously when speech is restored. In order to make this still more certain, the patient must be convinced that directly he speaks he will hear his own voice, and that he will then hear everything clearly. There is rarely any difficulty in curing the mutism by simple explanation and persuasion, though in our earlier cases we occasionally resorted to the suggestion produced by applying faradism to the larynx or making the patient excited with an anæsthetic. In the following two cases the latter method cured the deaf-mutism, but was followed by partial amnesia.

Hysterical deaf-mutism following shell-explosion: cured by suggestion with etherisation and followed by partial amnesia.—An Australian soldier, aged 22, wrote the following letter to his relations on August 21, 1916. "You may be a little surprised to hear that I am in the hospital suffering from shell-shock, which has taken away my speech and hearing. It is some sixteen days now since it happened. . . . We were in the trenches and going for dear life, when two of us spotted a German machine gunner in a hole, so we made up our minds to have him. We made a charge at him, and I just remember getting to him when a high-exposive shell burst at my head; it seemed as if it burst inside my head; everything went black. I tried to call out and couldn't, and

I could not hear my mates—only just a terrible bursting in my head all the time. I never remembered anything more until I came to on the boat. . . . The doctors have told me that I will get all right in time. . . . I saw a good deal of France. . . . There is not a young man there who is not in the Army. The girls and women work in the fie——"

The letter ended abruptly at this point, as I then came to examine him. The previous day I had hypnotised him without difficulty, but was unable to make any effective suggestions, as the deafness persisted during the hypnotic sleep, so that the suggestions did not reach the higher centres of his brain, and were consequently not acted upon either whilst he remained asleep or after he awoke. He was so deaf that he heard nothing at all during an exceptionally violent thunderstorm. He was not only unable to speak, but could make no sound of any kind and could not cough.

As no improvement had taken place, he was given ether, after being told in writing that it would have the effect of restoring his speech and hearing. He began to struggle after a first few whiffs, and long before he was anæsthetised he began to repeat the word "Mother," first in a whisper, then louder and louder until he shouted it with a stentorian voice that would have filled the Albert Hall. The etherisation was then discontinued, his limbs never having become relaxed. As he came round, I told him to say various words, which he repeated after me, and I then carried on a continuous conversation with him. When the effects of the anæsthetic finally passed away, he was talking with a normal voice and he had completely recovered his hearing.

His memory, which had previously been unaffected, was now a complete blank from a short time before he was blown up to the moment he had regained consciousness. He had no recollection of having lost his speech or hearing; he was astonished to see the letter he had begun, as he remembered none of the events described in it, and he did not remember having seen me before. In all other respects his memory was perfect.

In this case the patient became speechless from fright at the sound of the explosion, and deaf from the accompanying

noise; almost immediately afterwards he lost consciousness as a result of the aerial concussion. The moment he recovered consciousness the fact that he had lost his power of speech and hearing after the explosion recurred to his mind, and as a result of auto-suggestion these disabilities were perpetuated. The subconscious inhibition of speech and hearing was broken down as a result of the loss of control of the higher over the lower cerebral centres when he was under the influence of ether. By keeping these faculties continuously at work whilst the effects of the ether passed off, their recovery was maintained when consciousness returned.

Amnesia following recovery from deaf-mutism.—Sergt. H., a New Zealander, was admitted under my care on September 25, 1916. He was completely deaf and dumb, and wrote the following history of his case. "About September 17, our brigade was in support behind F——. A shell landed on a cookhouse, killing one and wounding two others, and as the Germans were bombarding very casually (about one shell a minute) I thought there was time to get those fellows in. I had just got to them—the cookhouse was fifty yards in rear of the trenches in the open—when the enemy opened battery fire and violently bombarded the sector with large shells. My man, whose both legs were broken, made progress very slow, and another shell landed within six feet of us, a piece of it hitting me in the back. I remember getting to the dressing station somehow or other, and next found myself in E—— four or five days after. I could not walk, talk, or hear. I have all other faculties and can understand and think clearly. I am just beginning to walk a few steps at a time. The M.O. at the hospital in France told me it was only a matter of time and I would be all right."

On October 1, as there was still no return of hearing or speech, he was given a little ether, and after a good deal of persuasion, his speech and hearing were restored. When he regained consciousness he could hear and speak perfectly, but he believed that it was May 25, 1915, and thought the "boys were kidding him" when they told him he was in England, as he had no recollection of having left New Zealand and imagined he must be in the Porarua lunatic

asylum. The following is an extract from an account he wrote of his doings on what he believed to be the previous day. "On May 24, I went to church for my organ lesson at 8 p.m., and afterwards had the treat of my life. Mr. F. played the pick of musical compositions until 11 p.m. That night will ever stand out as one of the most enjoyable of my life. . . . On Tuesday night I went to Mr. B.'s house to bid farewell to Wilf. and Rol., who are going to the war. I don't think things are so bad as to warrant my throwing up my billet, as yet, and I told them so. Besides, I would miss that examination. Of course, if the worst comes to the worst I will go to the front."

His general health rapidly improved, and in a few days, with encouragement and re-education, he learnt to walk quite well. But the blank in his memory persisted, and at the beginning of December, when he returned to duty, he was still unable to remember anything between May 25, 1915, and October 1, 1916, which appear to him to be consecutive days, and he had to learn his drill again, as he had no recollection of his military training.

Uncomplicated hysterical deafness is much more difficult to treat, and for a time our results were less satisfactory than in any other hysterical condition. For this reason we were induced to employ various forms of suggestion, although we realised that the methods were not really satisfactory. Suggestion under hypnosis was never of any use, as the patient remained deaf whilst hypnotised, and consequently heard none of the suggestions which were made to him. Electricity as a means of suggestion was sometimes successful, but often failed. In a number of cases of absolute bilateral deafness, which had resisted all other forms of treatment for some months, we performed "fake" operations, making a scratch behind the ear in the incompletely anæsthetised patient.

Hysterical deafness following exposure to a shell explosion cured by a fake operation.—Lance-Corporal M., 26 years old, was blown up by a shell on August 29, 1916. He became completely blind, deaf, and dumb, although he did not

lose consciousness. His sight returned the following day. On reaching England he was able to read and write, and he talked in his sleep. In spite of treatment with encouragement, electricity, and etherisation, no further improvement occurred until one night in November, when he woke up and asked the sister for a drink. After this he was able to talk normally, but the deafness remained.

He came under my care at Netley on March 21, 1917, seven months after the onset of the deafness. He was found to be completely deaf both to air and bone conduction, though he could feel the vibration of a tuning-fork on his mastoids. A loud noise just behind his head caused a slight tremor of his hands, blinking, and dilatation of the pupils, although he heard nothing; a slighter reaction was produced on the second and third occasion when the noise was repeated; after this it disappeared completely, and did not return again.

As the vestibular reactions were found to be unaffected, it seemed probable that the internal ear was free from organic changes. This was rendered still more likely by the fact that immediately after the explosion the deafness was associated with mutism, which is always hysterical when it develops after a shock of this kind. The patient was hypnotised by being made to stare at a lens for fifteen seconds, but the deafness persisted: he could not be made to respond to any suggestion, as he was unable to hear, and a loud noise produced no auditory-motor reflex, the pupils as well as the eyelids remaining fixed. The deafness also persisted during natural sleep, as it was found impossible to wake the patient by shouting "fire" and by banging a poker against a coal-scuttle within a few inches of his head, and no reflex flicker of his eyelids was observed. In the morning he had no recollection that anything unusual had occurred during the night. Suggestion with the aid of electricity, in which he had great belief, and attempts to re-educate the sense of hearing with various noises completely failed. He was, however, suddenly and completely cured by a "fake" operation on his ear on April 20, to his intense delight, as he had recently become extremely depressed

at the absence of any sign of improvement after more than seven months. His hearing was accurately tested the next day, and it was found that it was perfectly normal both to air and bone conduction, and the auditory-motor reflexes had returned. He was discharged to duty three weeks later, feeling perfectly fit. He visited the hospital on June 29, a few days before he returned to France; his hearing was normal, and he was well in every way.

This method of treatment was not invariably successful, and at the best it was not one which could be regarded as desirable, as it is much more satisfactory for the patient to understand the exact means by which he has been cured than for him to be fooled into a cure by gross suggestion. With increased understanding of the psychological basis of hysterical deafness we were able during the last year of the war to cure a large majority of cases by the most rational form of psychotherapy—explanation, persuasion, and re-education. The patient is made to understand by written explanations how he has become deaf, and how the original cause of his deafness has now disappeared: as the deafness was at first organic, he could not hear, however much he listened, and consequently after a time he ceased to listen at all. He is next persuaded to listen intently, and is taught that listening is just as active a process as moving, and requires a conscious effort on his part until it becomes automatic once more.

Even when a man has completely recovered his hearing, it may take some time before he becomes accustomed to the new conditions, especially if he has been deaf for a long period. When caught unawares he often fails to hear, though directly he perceives he is being spoken to he listens, and can then hear even with his eyes closed, so that lip-reading cannot help him. In severe cases he may hear the sound of the voice without being able to distinguish what is said. Re-education is then required in order that the sounds the patient hears, but at first cannot interpret, can be understood, that words which must at first be

repeated separately from each other can be used in continuous sentences, and that the voice which must at first be raised can become progressively more quiet. A similar method of re-education can be employed for the many people who are suffering from some incurable form of deafness, who can in this way be taught to make the most of what powers of hearing they still possess.

Similar re-education methods were used by Gordon Wilson [7] in the treatment of hysterical deafness in soldiers, and since these observations were made my attention has been drawn to a series of papers on the subject published in 1912 and 1913 by Maurice,[8] of Paris. He points out that in almost every form of deafness some improvement results from re-education. He has introduced a very costly noise machine or "kinesiphone" with the object of re-educating the power of hearing, but this does not appear to have any advantage over the simpler methods I have described. These have been used with success in a small number of very severe civilian cases, of which the following is an example.

Hysterical deafness after being struck by lightning; great improvement with psychotherapy nine years later.—Mr. S., aged 55, was struck by lightning in November 1911, when in New South Wales. His horse was killed under him, and he was left for dead by his companion who was riding with him. He was subsequently picked up and carried to a hospital, where he remained unconscious until the sixteenth day. A burn was found extending from the back of his head down the left side of his body to the hip. When he recovered consciousness he found that he had completely lost his hearing, and that his legs and arms were numb and partially paralysed. He slowly regained his power of movement, but continued to feel peculiar sensations in his limbs. The deafness was associated with a continuous noise in his head, which reminded him of escaping steam. It was so persistent that it often rendered it difficult for him to get to sleep.

When he left hospital he was told that his deafness was due to fracture of the base of the skull, caused by

the violence with which he struck the ground when he fell from his horse, as bleeding had occurred from his right ear.

When I first saw him in March 1920, I found that his vestibular reactions were perfectly normal. He could not hear a whisper at all; loud-spoken words were only heard within 2½ inches from the left ear and one inch from the right. Low notes were heard better than high ones. There was no paralysis, but he complained of inability to walk more than a short distance, and he was still troubled with unpleasant sensations in his limbs. I could find no evidence of organic disease, and regarded the deafness as probably hysterical in origin. This opinion was confirmed by Mr. W. M. Mollison, who could find no evidence of disease in either ear.

I explained to him that the terrific noise of the thunder-clap had produced temporary organic deafness, and that this was the last impression he had before he lost consciousness. It was quite impossible that the deafness was due to a fracture of the base of the skull injuring the auditory nerves, as he had been told, as in that case the vestibular nerves would have been involved at the same time. I pointed out that having once been deafened he had ceased to listen, and that when the temporary changes produced by the noise had disappeared he could have heard again, but he had become so impressed by the fact that he was deaf that he had not tried to listen. He at once improved when he realised that in order to hear he had to listen actively. I gave him a few lessons myself, and he then continued to train himself whilst at home in Scotland. On the railway journey he found that he could hear what other people were saying in spite of the noise of the train, although he had never been able to do this before, and when he got home he found he could hear the voices of his relations which he had not heard since the accident. He trained himself to listen to everything that was happening around him, and for the first time since 1911 he could hear the singing of birds, footsteps in his neighbourhood, and clocks striking.

Although in the past it had been impossible for anybody to wake him by noises, he now found that quite slight noises might wake him in the night, so that whatever the basis of the hysterical deafness might be, it clearly persisted during sleep, but was now no longer present. When I last saw him on June 16th, he could carry on an ordinary conversation without difficulty. Mr. Mollison found that with the right ear, which had been previously almost totally deaf, he could now hear clearly spoken words at a distance of eighteen inches, and with the left ear he could hear quietly spoken words six feet away and a whisper at one inch. He himself has noticed that even when his better ear is covered, he can carry on a conversation if the words are spoken slowly and clearly. At the same time his general health has greatly improved, and the noises in his head have almost disappeared. Whilst he learnt to give greater attention to sounds, he gave less attention to sensations coming from other parts of his body, and consequently the paræsthesia of his limbs gradually disappeared.

In the following case, that of a thirteen-year-old deaf-mute, who had already with great difficulty learnt to talk, remarkable improvement in hearing has occurred as a result of psychotherapy with the aid of some physical treatment, which by itself could have had but little effect.

Absolute functional deafness dating from infancy associated with slight organic middle-ear deafness.—Bertha M., aged 13, was seen by Mr. W. M. Mollison and me on March 8, 1920. As far as her parents could tell, she had never heard anything, but it was not recognised with certainty that she was completely deaf until she was two. Her adenoids were then removed, but no improvement occurred. Her parents are not related, and the only other member of the family who is deaf is her nine-year-old brother. In his case there was no indication of middle-ear disease and the vestibular reactions were almost completely absent, so we made no attempt to treat him by physical or psychical methods, as we regarded the prognosis as hopeless. Both children had the typical speech of deaf-mutes. Mr. Mollison found that the girl's

left membrane was dull and somewhat retracted, the right being more normal in appearance. The tonsils were buried and remains of adenoids were still present. A loud shout could not be heard, and no tuning-fork could be heard either through air or bone. There was some doubt as to whether she could faintly hear a high note produced by the monocord (4·6 cm.). The auditory-motor reflex was absent. Rotation and syringing with cold water gave normal vestibular reactions, but the giddiness and nystagmus were slight in degree.

Owing to the presence of vestibular reactions it was thought that the condition might prove to be wholly or in part hysterical. At the same time it seemed likely that the best results would be obtained by removing as far as possible the very slight physical abnormalities which were still present, as quite apart from the direct effects which might result therefrom, the operation might hasten improvement by its suggestive influence.

On April 4, 1920, Mr. Mollison inflated the Eustachian tubes under a general anæsthetic; the tonsils were enucleated and the adenoids curetted. A week later, when the first treatment by re-education was given, she was still completely deaf. In simple language I explained to her that listening was an active process and that she must no longer maintain a passive attitude towards external sounds as she had done in the past. At the end of the lesson, for the first time in her life, she could hear a little. I showed her mother and governess how to continue with the education, and when I saw her again on July 10, 1920, the improvement was most remarkable. It was now possible to carry on a conversation with her by talking loudly into her ear, and the day before her governess had told her a complete fairy story in this way. She is now having all her ordinary lessons by ear in addition to the special lessons in listening, and there seems every prospect of her ultimately hearing almost normally.

In this case naso-pharyngeal infection had apparently led to severe middle-ear catarrh in early infancy, and the resulting organic deafness had been perpetuated and exaggerated by

the interruption in the normal process of education in listening to and interpreting sounds.

[1] C. S. Sherrington and A. Forbes, *Amer. Journ. of Physiology*, xxv. 367, 1914.

[2] A. F. Hurst and E. A. Peters, *Lancet*, ii. 517, 1917; and A. F. Hurst, *Seale Hayne Neurological Studies*, i. 279, 1919.

[3] P. Briquet, *Traité de l'Hystérie*, p. 295, 1859.

[4] A. F. Hurst, J. L. M. Symns, and R. Gainsborough, *Review of Neurology and Psychiatry*, i., 1918; and *Seale Hayne Neurological Studies*, i. 19, 1918.

[5] J. S. Fraser, *Lancet*, ii. 872, 1917.

[6] D. E. J. Moure and R. Pietri, *Rev. de Laryngologie, d'Otologie, et de Rhinologie*, xxxviii. 153, 1917.

[7] J. L. Gordon Wilson, *Brit. Med. Journ.*, i. 867, 1917.

[8] G. A. Maurice, *Treatment of Deafness by Re-education of the Powers of Hearing*, Paris, 1913.

CHAPTER IX

HYSTERICAL HYPERACEUSIS

THE nervous individual, who has a brisk auditory-motor reflex which results in "jumping" at the least sound, is familiar in civil life. An exaggeration of this condition was a common symptom in soldiers suffering from all forms of war neurosis which were emotional in origin. In its most marked form the patient jumped violently with sudden sounds, which could hardly be heard at all by an ordinary individual, and louder sounds produced violent universal tremors. The condition persisted during sleep, the patient showing the same exaggerated response to sounds, which did not necessarily wake him, although they generally slept more lightly than they had done before the war. The type of sound which produced this reaction was always one which was in some way reminiscent of shell explosions or other sounds of battle. Many patients realised this quite clearly in their waking state. In one severe case of the kind, the patient, when deeply hypnotised, told me that he was always listening for shells, and jumped whenever a sound reminded him of them, although he had been unable to explain the symptom when he was awake.

I have already described the experimental and clinical observations which prove that the auditory-motor reflex is a mid-brain phenomenon. I believe that its exaggeration in certain neuroses can be readily explained as a result of a mechanism exactly the reverse of that which leads to its abolition in hysterical deafness. Whereas in hysterical deafness the patient does not listen, a soldier who is in a state of constant terror becomes accustomed to listen for shells with abnormal concentration, and this concentration

may persist when he is no longer at the front and no shells are bursting. Instead of the resistance at the synapses in the auditory tract being increased as in hysterical deafness, it is diminished owing to the extreme degree with which the dendrites are extended. This results in an abnormally brisk auditory-motor reflex and probably also in abnormally acute hearing. We only tested the power of hearing accurately in one patient, in whom the jump reflex was excessively developed. Captain E. A. Peters and I found that he could hear sounds at a distance four times as great as the average individual, which means that his power of hearing was sixteen times greater than the average, as the intensity of sound diminishes inversely as the square of the distance. His acuity of hearing was most remarkable. Sentences whispered in one corner of a large room so quietly that a group of officers in the centre heard no sound at all were correctly repeated by him, although he was sitting in the opposite corner. The hyperaceusis and jump reflex were quite uninfluenced by the administration of one hundred grains of bromide a day, and were only slightly reduced by plugging the ears with plasticine.

The increased resistance at the synapses in hysterical deafness persists during sleep. In the same way the diminished resistance in hyperaceusis persists, as the patients wake with unusual ease in response to sounds, and also show a brisk jump reflex even when they do not wake.

CHAPTER X

SEEING, LOOKING, AND HYSTERICAL BLINDNESS

In Chapter VIII the psychology of hearing was discussed. It was shown how in order to hear it was necessary to listen, and that the process of listening involved some change at each synapsis in the auditory tract from the cochlea to the cerebral cortex, as a result of which resistance to auditory impulses was diminished.

In order to see it is necessary to look. Looking involves a process in the visual tracts strictly comparable to what occurs in the auditory tracts in listening. But, in addition to this, the action of a number of muscles is called into play, comparable to what I described in regard to listening, although the movements involved are more important and require much more delicate adjustment. Two distinct elements thus require consideration—the afferent, involving the visual tracts, and the efferent, involving certain motor tracts.

(*a*) **The Afferent Element.**—Inattention is rarely so profound in normal individuals that no visual impulses reach the brain at all, although complete absence of hearing not infrequently results from extreme inattention. It is common enough for visual impulses to be so blurred that a man, wrapped up in his own thoughts, does not see the approach of a friend, but he would always avoid obstacles if he were walking, and he would be aroused by the sudden approach of an object to his eye; the latter would also give rise to the flinch reflex.

In the condition of stupor, which was not uncommon in soldiers who had been exposed to exceptionally terrifying ordeals, the mind appeared to be so completely absorbed

with thoughts which had no connection with the patient's present surroundings that he did not respond to any impulses from the outside world. He appeared to be blind, deaf, and anæsthetic; he gave no flinch (visual-motor) or jump (auditory-motor) reflex, cutaneous (tactile-motor) reflexes were often though not invariably abolished, and the pupil contracted sluggishly or not at all on exposure to light. In spite of this he could feed himself if food were put in front of him, and did not stumble against obstructions if he were taken for a walk, and he occasionally showed a slight response to certain loud sounds, so that vision and hearing were clearly still possible; but it was difficult or impossible to induce the patient to look or listen even for a few seconds at a time. Exactly the same thing occurs in somnambulism. The doctor, seeing Lady Macbeth walking in her sleep, exclaims, "You see, her eyes are open," and the gentleman replies, "Ay, but their sense is shut."

In the chapter on "Hearing, Listening, and Hysterical Deafness," it was explained how the idea of being unable to hear, suggested by temporary organic deafness, might give rise to a continued absence of listening and consequent deafness after the organic cause had disappeared. In the same way any condition which has led to complete though temporary blindness may suggest to the individual that he has lost his sight for ever: this is particularly likely to be the case if the temporary blindness is produced suddenly under terrifying conditions, as, for example, by the explosion of a powerful shell in the immediate neighbourhood. The slower onset of the temporary blindness in gassing, although the surrounding conditions might be equally terrifying, generally resulted in less profound hysterical blindness. When the suggestion that the sight is permanently lost has become thoroughly accepted, the individual will cease to look. The visual tract is no longer prepared for sight by attention, and visual impulses consequently cease to pass up to the brain. In the act of looking the resistance at each cell-station in the tract is diminished by some such process as a throwing out of dendrites or an alteration in the electro-chemical condition of the synapses.

Consequently visual impulses not only give rise to no visual perception, but the flinch reflex and in the most severe cases even the pupil reflex to light disappear, as the resistance to the impulses, even at the lowest synapses, is too great to be overcome. More commonly the impulses produced by a very bright light can still break through the resistance, so that the light is perceived and a sluggish reflex to light is obtainable, but the flinch reflex is still completely abolished.

When, as a result of psychotherapy, the patient realises that he can really see if he only chooses to look, he once more throws out the dendrites or otherwise reduces the resistance in the visual path, and vision returns, the pupil and flinch reflexes becoming normal again at the same moment.

Injury or disease of the visual centre in the occipital cortex is the only organic condition leading to blindness in which the lower visual centres are unaffected. It might be expected that the flinch reflex would remain unaltered, corresponding with the persistence of the auditory-motor reflex in decerebrate animals. This is not, however, the case, as Gordon Holmes, [1] in his unique experience of head injuries during the war, found that the flinch reflex was always lost in the blind fields of vision. This corresponds with the experimental observations of Sherrington, who found that the reflex was always abolished in decerebrate animals, even when the anterior corpora quadrigemina remained intact, although the auditory-motor jump reflex remained unaffected. It is not at first clear why the latter should persist while the flinch reflex should disappear. But Sherrington points out that it is not every sound which can produce the auditory-motor reflex in decerebrate animals, but that shrill peculiar sounds are the provocative ones, mere loudness appearing to be of no importance. As the effective sounds are neither injurious nor painful, they must have some other meaning, possibly connected with the sexual life of the animal. On the other hand, the retinal stimulus which gives rise to the flinch reflex may perhaps require remembered experience in order to be

effective. Thus the reaction of blinking is anticipatory, the response being to threatened injury and not to actual injury. As memory and anticipation depend upon higher centres than the corpora quadrigemina and geniculata, the reflexes are abolished both in animals and man when the cortical centres are thrown out of action. The flinch reflex is thus a true visual reflex, being dependent, as von Monakow [3] was the first to show, on visual perceptions. Yet how instinctive a reaction it is to a threatened blow is shown by Darwin's observation that he could not repress his blink when a snake on the other side of the glass of the snake cage at the Zoo struck at him.

It is clear, from what has been said, that in the absence of abnormality in the eyes there is no means of determining with certainty whether absolute blindness in the whole fields or in homonymous areas is organic or hysterical, as the flinch reflex is lost in both.

(*b*) **The Efferent Element.**—With the afferent element set in readiness, visual impulses reach the occipital lobes, but nothing is seen clearly until the eyes are opened, the extrinsic muscles work in thorough co-ordination, so that the object to be looked at is brought into the centre of the field of vision of both eyes, and the ciliary muscles contract just sufficiently to bring it into correct focus. All these motor processes may be impaired as a result of suggestion, either alone or in addition to the afferent element already described. How this occurs can best be understood by a consideration of the hysterical disorders of vision which follow gassing.

Exposure to mustard gas is followed in a few hours by pain in the eyes, which is increased by exposure to light. Conjunctivitis, blepharitis, and in rare cases keratitis, quickly develop. As long as pain and photophobia are present, the swollen lids remain closed. This is partly due to inactivity of the levator palpebræ superioris muscles, for the patient makes no effort to open his eyes, knowing that if he were to do so the exposure to light would cause pain. It is partly due to a protective reflex, which results in over-action of the orbicularis palpebrarum muscles, particularly

if the patient does try to open his eyes. The object of this is to protect the eyes from being irritated by light; it is accompanied by reflex lachrymation, as a result of which irritating material is washed away. The inflammation gradually subsides, the reflex blepharospasm and lachrymation disappearing *pari passu*. At the same time the majority of patients realise that they can now open their eyes without hurting them, and in most cases they do so.

In individuals, however, who have become abnormally suggestible as a result of the stress and strain of active service, and in others who for any reason are particularly anxious about the condition of their eyes, the normal results of the conjunctivitis become perpetuated by auto-suggestion, to which may sometimes be added the hetero-suggestion caused by injudicious treatment, such as the prolonged use of eye-shades and dark glasses, which give rise to an exaggerated fear in the patient's mind of the consequences of having been gassed. The voluntary inactivity of the levator palpebræ superioris becomes perpetuated as an involuntary inactivity, which may amount to actual paralysis, the condition being now one of hysterical ptosis. In rare cases the hysterical paralysis of the levator palpebræ may spread, hysterical paralysis of the whole of the face resulting (case 8). A patient who finds himself unable to open his eyes by contracting his levator palpebræ muscles often attempts to compensate for this by contracting his frontalis muscles, and if the attempt meets with sufficient success to enable him to see through the chinks between his eyelids, he does this more or less constantly. The reflex blepharospasm is perpetuated as hysterical blepharospasm, which is generally most obvious when the patient attempts to open his eyes voluntarily, the eyes becoming more tightly closed than ever. The paralysis of the levator is thus accompanied by spasm of the orbicularis, just as hysterical paralysis of one group of muscles in a limb is often accompanied by hysterical spasm of the opposing group of muscles. The spasm often involves the neighbouring muscles or even all the muscles supplied by the facial nerve. Any attempt

to pull the eyes open meets with great resistance, and a peculiar thrill is often felt owing to the irregular contraction of the orbicularis muscle. In some cases, after it has become possible to open the eyes voluntarily, the ptosis and blepharospasm manifest themselves intermittently in the form of attacks of blinking, especially on exposure to a bright light. Although hysterical ptosis and hysterical blepharospasm are generally present together, either may be present alone, and the proportion of one to the other may be different in the two eyes, as in case 8.

In total blindness due to severe bilateral optic atrophy the eyes are kept open during the day, and look straight forward, but they close during sleep. An uneducated man, however, if told to pretend that he is blind, generally closes his eyes, and when they are forced open, or he is told to open them, he at once turns them upwards in order to keep the pupils covered by the lids. Conversely, if he is for any reason unable to open his eyes, he will very likely imagine that he is blind. Hysterical ptosis and blepharospasm are thus often accompanied by hysterical amblyopia, the patient having so convinced himself that he cannot see that he makes no attempt to look when his eyes are at last opened. When the lids are forced apart, the eyes generally turn involuntarily upwards to protect them from the light. The pupils are consequently still hidden, and vision is impossible. Even when the lids are sufficiently separated to expose the pupils, the patient can only see indistinctly, and in rare cases he cannot see at all. The indistinct vision is due to a combination of hysterical paresis and spasm of accommodation. A man with normal vision relaxes his ciliary muscles to look at the distance, and contracts them to look at a near object. A man who is convinced that he cannot see, fails to regulate the activity of his ciliary muscles correctly when he opens his eyes. Instead of relaxing them when he wishes to look at a distant object, he throws them into spasm, and he also fails to see near objects clearly, as he does not contract them to the correct extent. He has, in fact, lost control over accommodation, and, believing himself blind, looks at nothing.

The external ocular muscles may remain inactive as hysterical external ophthalmoplegia—a very rare condition, which I have, however, seen occasionally—or, much more frequently, certain muscles may be thrown into spasm in the attempt to bring them into action after long disuse, and hysterical strabismus results. Thus hysterical spasm of convergence is often seen when the eyelids are forced open, in addition to the contraction of the superior recti, which tends to keep the pupils hidden under the upper lids. In consequence of these abnormalities of accommodation and convergence, the patient sees nothing clearly, but he is not completely blind. Complete hysterical blindness, which is rare in cases of this kind, is due to the patient being so convinced that he cannot see that he does not use his visual centres at all; the psychology of this condition has already been described.

The following cases of hysterical disorders of vision have been selected from the large number I have seen as illustrating what I have already said concerning their pathogenesis. They also throw light on several questions connected with the ocular movements and reflexes, which have been the subject of controversy in the past. For the description of the majority of cases I am indebted to the medical officers who worked with me at the Seale Hayne Hospital, especially Captain A. Wilson Gill, Captain C. H. Ripman, and Captain R. G. Gordon.[3]

In the first case the blindness was the most nearly absolute I have seen result from hysteria. It is of exceptional interest, as it is also the only case in which the reflex contraction of the pupils to light was abolished. So far as I am aware no similar case has hitherto been described.

Case 1.—*Total blindness with loss of pupil reflexes following shell-concussion: hysterical nature proved by rapid cure by psychotherapy after persisting for four years.*—Pioneer B., aged 41, went to France in September 1914. After six weeks' fighting he was stunned as the result of the explosion of a shell in his immediate vicinity. In the evening he noticed he could not see clearly, and attributed this to the

shock of the explosion. He at once feared he would lose his sight: his eyelids began to droop, and he had difficulty in focussing near objects. He was sent to England, where the use of eye-drops and dark glasses confirmed his fears, and in a short time he became totally blind. Early in 1915 he was discharged as permanently unfit, receiving a full pension for total blindness. He was examined every six months after this, but no treatment was given. In November 1918 he was seen by Mr. J. R. Rolston, of Plymouth, who recognised the condition as hysterical, and advised his transfer to Seale Hayne Hospital, where he was admitted on November 13, 1918. He presented the picture of the typical blind beggar of the street. Unshaven, unkempt, and dirty, and wearing a pair of dark glasses, he came supported by his wife, while in his hand he carried a thick stick to help to guide himself. Whilst wearing the glasses he kept his eyes open, but could see nothing; when they were removed he was unable to raise the lids owing to severe blepharospasm, except in a darkened room.

Treatment was commenced the same evening, and in a short time the blepharospasm was overcome, and the patient opened his eyes, but he was still totally blind. *The pupils were then found to be widely dilated, with no trace of reaction to light.* The flinch reflex was completely absent in both eyes. Ophthalmoscopic examination showed nothing abnormal, and a definite diagnosis of hysterical blindness was made. Explanation as to the nature of his condition and encouragement to use his eyes rapidly led to partial restoration of vision, but at the end of two hours he still stumbled over objects placed in his path. After a rest of an hour, treatment was continued and further slight improvement occurred. In attempting to focus his eyes he made strong contractions of the muscles of the neck, similar to those seen in the spastic variety of hysterical aphonia when the patient attempts to speak.

The next morning he was taken out of doors, and distant objects were soon recognised. An endeavour was then made to teach him to focus his eyes on nearer objects, and by the evening he could read 6/24 at 20 feet. The excessive

contractions of the neck muscles continued, however, but by encouraging him to relax they gradually disappeared, and vision steadily improved. He was still inclined to stumble over objects placed in his path, but this was merely due to inattention. On November 25 he could read with each eye in turn 6/12 at 20 feet, and he could spell words printed in small type, but as he was almost completely illiterate he could not pronounce them. The flinch reflex and the normal pupillary reactions to light had returned the first evening.

The patient was also completely deaf in the left ear on admission; he was given instruction in listening, and at the end of a week he could hear normally. When seen four months later, in February 1919, he was at work as a watchmaker and gramophone repairer.

Whilst a man is still dazed as a result of being blown up by a high-explosive shell, he pays no attention to any external stimuli, and may be regarded as psychically blind, deaf, and anæsthetic. His sight, hearing, and cutaneous sensibility generally return as he regains consciousness, but if there is anything which draws his attention to his eyes, the blindness may persist as a result of auto-suggestion. The following is one of seven cases seen with Major A. W. Ormond,[4] in which sand was blown into the men's eyes from the sand-bags of the parapet on which the shell exploded. The irritation it caused drew their attention to their eyes, and resulted in blepharospasm and amblyopia, which persisted long after every sign of conjunctivitis had disappeared. These early cases were the only ones in which hypnotism was used.

Case 2.—*Hysterical blindness following exposure to a shell explosion cured by hypnotism.*—The patient, aged 22, was looking over a parapet at Gallipoli on July 18, 1915, when a shell struck the sand-bags in front of him. He remembers the sand being thrown up into his eyes, after which he fell back and knocked his head. He was unconscious for twenty-four hours. His first impression on regaining consciousness was extreme irritation in his eyes. He tried

to open them, but found he could not do so. His mind thus became concentrated on his eyes, and owing to the confusion which is common among the uneducated between inability to open the eyes and blindness, he became absorbed with the idea that he was blind, and that he would never be able to open his eyes or see again. The impairment of other functions, which was doubtless present at first, remained unnoticed in this greater trouble, except for some loss of hearing, which quickly disappeared. The condition of his eyes had not altered when I first saw him on September 17, 1915. He was quite blind, and there was a constant flicker of his eyelids, which were kept almost closed. On forcibly opening his eyes they were found to be turned so far upwards that it was difficult to see even the iris. A few fragments of sand were still embedded in the conjunctiva, but not in the cornea; there was no inflammation. The inability to open the eyes and the idea of blindness were thus perpetuated by auto-suggestion, and persisted long after the inflammation caused by the dust had disappeared.

The patient was easily hypnotised, and whilst asleep he was told that he would be able to see when he woke up. The moment he awoke the suggestion was repeated very forcibly, and his eyes were held open. He cried out that he could see, tears ran down his cheeks, and he fell on his knees in gratitude, as he had thought that he was permanently blind, and believed that his sight had been restored by a miracle. When seen again on September 20, the external appearance of his eyes was normal, and he said that he was able to see as well as he had ever done. There was some opacity of the vitreous of the left eye, which was doubtless a result of injury at the time of the explosion. There had been no return of symptoms, and the patient was well in every way when I last saw him on September 30. His vision was 6/6 in the right eye and 6/36 in the left.

In the next case the blindness was so typical of what would be expected to follow a wound in the occipital region that no doubt was felt as to its organic nature. The sequel, however, proved that it was due to the perpetuation

by auto-suggestion of the organic blindness, which had been caused by concussion rather than destruction of the visual centres in the occipital cortex. The patient, realising that he was blind in certain directions, ceased to look in these directions; the dendrites were perhaps retracted at the synapses in the affected tracts, and the flinch reflex consequently disappeared. When at last the œdema and other changes produced by the initial concussion of the occipital cortex had disappeared and vision was again possible, the patient had become so accustomed not to look in the blind fields that no spontaneous improvement occurred.

Case 3.—*Hysterical blindness in three quadrants of the visual field, following organic blindness caused by a wound in the occipital region, and associated with hysterical deafness.*—Private W., aged 22, was wounded over the lower part of the right occipital region near the middle line on June 7, 1917. He was unconscious for five days, and was then trephined. On admission to Netley on July 6, 1917, he was completely deaf in both ears, but as the vestibular reactions on rotation were normal, the deafness was regarded as hysterical. It was noticed that he had difficulty in seeing, and that he held anything he wished to read low down on the right side, although he volunteered no complaint about this, and only spoke about his deafness. On further examination it was found that he was totally blind except in the right lower quadrant of the field of vision of both eyes, the blindness being what might be expected to result from the wound, which had probably involved the whole of the visual centre on the right side, but only the lower part of the calcarine cortex on the left. An attempt was made at the end of August to cure the hysterical deafness by a pseudo-operation, the patient being told that a cut behind his ear would certainly restore his hearing. Nothing was said to him about his blindness, which was regarded as organic. The "operation" resulted in immediate improvement in his hearing, as it at once became possible to carry on a conversation with him by shouting. Quite unexpectedly it was found that his vision was now absolutely

normal, the blindness having been cured by the suggestive effect of the "operation."

I shall now describe the different varieties of hysterical visual disturbances, which were such a frequent result of gassing. The first case is an example of the common form in which both eyes were equally affected; the patient was unable to open his eyes owing to a combination of hysterical ptosis and blepharospasm, and when the eyes were opened he only saw very indistinctly.

Case 4.—Hysterical blepharospasm, ptosis, and amblyopia of four months' duration.—Gunner K., aged 33, had been in France seven months, and for most of the time was on duty behind the firing line, because his nerves were not equal to life at the front. In December 1917 he was blown up, and was then very shaky and frightened. He was gassed on April 9, 1918. Next morning he was unable to open his eyes. When admitted to Seale Hayne Hospital, under Captain C. H. Ripman, in August 1918, he could only see indistinctly through the narrow slit which he could produce by a great effort between his eyelids by strong contraction of his frontalis muscles, and he used his hands to steer himself. A thrill caused by contraction of the orbicularis muscles was distinctly felt in the lids when an attempt was made to force them open (fig. 20).

The orbicularis spasm relaxed, and he recovered the power in his levator palpebræ muscles in five minutes as a result of persuasion, and he was then quickly trained to see perfectly well. For a time he was inclined to blink and to let the upper eyelids droop a little; this was due to persistence of slight ptosis, caused by hysterical paresis of the levator palpebræ muscles, and not to spasm of the orbicularis, and in order to counteract the ptosis he continued to wrinkle his forehead. He made a slow but steady recovery from this habit, and was discharged from hospital completely cured a few weeks later.

In cases 5 and 6 one eye was more severely affected than the other. There must always be some explanation for

an asymmetrical condition resulting from a cause which would be expected to act symmetrically. In hysteria the explanation is always a psychical one. In the cases in which visual symptoms were more marked in one eye than the other after gassing it was found that the patient had for some reason been anxious about the vision of the former, which may, in fact, have been less acute than the other on account of an error of refraction.

Case 5.—Hysterical ptosis with unilateral blepharospasm and amblyopia cured in an hour.—Corporal B. was gassed in France in June 1918. This resulted in severe conjunctivitis. As he could not open his eyes, he was afraid he might go blind. He was particularly anxious about his left eye, as it had always been weak and subject to inflammation and styes.

In order to protect the eyes from the pain caused by exposure to light he had kept the lids closed, and when the inflammation had subsided he found he was unable to open them. If the left lid was raised, he experienced great discomfort, and everything seemed blurred. The right eye was not painful, and he could see clearly with it; but in order to do so he had to raise the lid by contracting the frontalis muscle, as he could not use his levator palpebræ (fig. 21 (*a*)).

He was admitted to Seale Hayne Hospital, under Captain R. G. Gordon, on October 23, and an hour's persuasion was sufficient to induce him to use the proper muscles and to relax the spasm of the right frontalis (fig. 21 (*b*)), but the spasm tended to relapse for a few days unless he paid special attention to it. The vision of the left eye quickly returned with re-education of accommodation.

The next case is of interest in connection with the part taken by the frontalis muscle in ptosis. It is generally taught that hysterical ptosis can be distinguished from ptosis due to organic disease by the fact that the latter is accompanied by compensatory over-action of the frontalis muscle in the effort to keep the eye open, whereas this never occurs in the former condition. Thus Purves

Stewart [5] and Oppenheim [6] make the distinction without mentioning the possibility of exceptions. In the following case, however, and also in cases 4, 5, and 7, there was as marked compensatory over-action of the frontalis (figs. 20, 21 (*a*), 22 (*a*), and 23 (*c*)) as occurs in any case of organic paralysis. The complete recovery after a few minutes' treatment by psychotherapy, although the ptosis had persisted without alteration for a considerable time, confirmed the diagnosis of hysteria. It is not surprising that the belief that compensatory action of the frontalis only occurs in organic ptosis should prove erroneous, as it would seem very natural that a man, who was unable to open his eye by contracting the levator palpebræ superioris, should attempt to do so with his frontalis muscle, and it is obvious that in organic ptosis the over-action of the frontalis is purely voluntary. As the exact manner in which hysterical ptosis manifests itself depends simply on the patient's own conception of a drooping eyelid, it would be astonishing if the frontalis did not always contract, and in all the cases of pure hysterical ptosis which I have examined the frontalis did in fact contract. It is clear, therefore, that the supposed distinction between organic and hysterical ptosis, which has been copied from one book to another, cannot be regarded as of any value in diagnosis.

Case 6.—*Hysterical blepharospasm, ptosis associated with over-action of the frontalis, and amblyopia, following gassing and cured by psychotherapy.*—Corporal H., aged 22, was gassed on May 20, 1918. He was admitted to hospital the same day with conjunctivitis, his eyes being tightly closed. Thirteen days later he was able to open his left eye, but the right one still remained closed, and he thought he was blind in that eye. Early in June he was transferred to a military hospital in England, where he remained for a fortnight. He was then sent to a V.A.D. hospital, and treated with electricity and daily eye-baths with no improvement. He was admitted to Seale Hayne Hospital on October 22, under Captain S. H. Wilkinson, with the right eye tightly closed owing to unopposed spasm of the orbicularis, but the left eye was kept partially open as a

result of continuous contraction of the frontalis muscle (fig. 22 (*a*)). Vision was so indistinct that he could only see with difficulty, and he was quite unable to read. No conjunctivitis was present, but at the corner of the right eye there was a scar on the skin about the size of a two-shilling piece. This was caused by a nævus having been burnt off when he was a child; he had always believed that this had impaired the sight of his right eye. After being in France for a few months, he noticed that the sight of the right eye was becoming more "blurred," and when he was gassed he was at once terrified that he would be completely blinded in this eye. He was treated by explanation and persuasion, and in half an hour the orbicularis spasm of the right side and the ptosis of the left were overcome (fig. 22 (*b*)). He was then quickly trained to focus his eyes, so that his vision became quite normal except for some myopia of the right eye, which had always been present, and was doubtless the real cause of this eye being the weaker one.

In the following case the ptosis which was present on one side spread so as to produce facial paralysis, which was complete except for the frontalis, which attempted to counteract the paralysis of the levator palpebræ superioris. The platysma was also involved, contrary to what would be expected from Babinski's teaching, as he regards paralysis of this muscle in facial paralysis as a definite sign that the condition is organic. This is not, however, the only case I have seen in which Babinski's platysma sign has proved to be misleading. Whilst the ptosis spread to produce facial paralysis, the blepharospasm of the affected side spread to produce facial spasm.

Case 7.—Hysterical left facial paralysis and ptosis, right facial spasm, paralysis of right arm and both legs, aphonia and dysarthria, and amblyopia, following gassing.—Lieutenant B., aged 28, was gassed on April 24, 1918. He remained quite blind and very hoarse for about six days. When sent to England on May 3 he could see a little, and could talk fairly well. On reaching the hospital, however, he could

hardly open his eyes, and he was very breathless. The base of the right lung was dull, and crepitations were present; his sputum was blood-stained. He improved, but the left side of his face and his right arm and leg became paralysed during the next few days. He gradually lost the power of speech; he could not phonate at all, and could only make unintelligible, whispering sounds. He was unable to open his mouth, and could thus only take fluid food. A few days later the left leg became swollen, and a tender spot developed over the femoral vein, apparently due to thrombosis. The other leg soon became affected in the same way to a less extent, but rapid improvement occurred, so that the last trace of swelling had disappeared by June 10.

When I first saw him on June 16 his eyes were closed, and there was a constant spasm of the whole of the right side of the face, especially involving the orbicularis palpebrarum and levator anguli oris (fig. 23 (*a*)). The left side of the face, including the platysma, was completely paralysed, except for the frontalis, which contracted with excessive vigour in the patient's efforts to overcome the ptosis (fig. 23 (*c*)), which was present on both sides in addition to the spasm on the right side, and prevented him from seeing unless he held his left eye open with his fingers (fig. 23 (*b*)). When the left upper lid was held open and the right lids pulled apart, it was found that there was a marked squint due to spasm of the internal recti. The masseters were tightly contracted, so that he could not open his mouth. Spasm was also present in the left side of the tongue, so that when later he was able to open his mouth and put out his tongue it went to the left. The right arm and leg were completely paralysed, and he was only able to move the left leg with difficulty. With simple persuasion and re-education in breathing he quickly learnt to phonate, and then gradually learnt to articulate clearly. The ptosis of the left eye was overcome by persuasion without difficulty. The spasm of the muscles of the right side of the face slowly improved with massage and stretching the muscles by pulling the eyelids apart and

(*a*) Position at rest, showing double ptosis, right-sided facial spasm and left-sided facial paralysis.

(*b*) Same as (*a*) with left eyelid raised so as to see. This could not be done with the right eye owing to spasm.

(*c*) Voluntary effort to open eyes, resulting in over-action of left frontalis although left side of face is paralysed, and spasm of right side of face including platysma.

FIG. 23.—Hysterical ptosis of left eye and paralysis of left side of face with hysterical spasm of right side of face.

pulling down the upper lip, which was gripped with one finger inside the mouth and the other outside. When at last he was able to open the right eye it was found that he could hardly see with it, but with simple explanation and persuasion the amblyopia disappeared. The severe internal strabismus, which was present when both eyes were open, gradually disappeared as he became accustomed to using his eyes. With some difficulty he was taught to balance himself whilst standing, after which he quickly learnt to walk. He was treated at intervals from 10 a.m. until 6.30 p.m., by which time he could walk normally. His right arm had now recovered without special treatment, and for the first time since the onset of symptoms he was able to write. He could talk with a normal voice, but in a somewhat laboured manner. The paralysis of the left side of the face and the ptosis had completely disappeared, but there was still some slight spasm of the right side, although he could now open the eye and see quite clearly, and there was no squint.

It is difficult to account for the extraordinary variety of hysterical symptoms which developed in this officer; but from his history it is apparent that whilst some were produced by auto-suggestion, others probably developed as a result of unconscious suggestion on the part of those who examined him. He must have been constitutionally suggestible, and the stress and strain of active service, and particularly a heavy bombardment with gas shells every night before he was finally gassed, must have rendered him still more suggestible. As was so often the case in the war, most of the hysterical symptoms developed gradually during the stages of his journey to the base and thence to England, whilst some only appeared after his arrival at an English hospital. The reflex protective aphonia, which resulted from the irritating action of the gas on his throat, was perpetuated by auto-suggestion. The pulmonary thrombosis, which appears to have developed on his way to England, led to further respiratory symptoms, which were exaggerated and perpetuated by auto-suggestion, so that to the original aphonia was added a severe dysarthria.

Frequent examinations of his nervous system must have unconsciously given rise to the idea in his mind of the paralyses and spasms affecting various parts of his body, and the original protective closure of his eyes was perpetuated as blepharospasm on the right side and ptosis on the left, the former spreading so as to involve the whole of the side of the face and the latter becoming complicated by left-sided facial paralysis. The closure of the eyes led by auto-suggestion to the idea of blindness.

As his hospital was twenty miles away I had only one other opportunity of treating him before his discharge, but in spite of this he remained fit except for an attack of phlebitis and a periodic left-sided headache, which made him wonder whether he was not still suffering from organic brain disease. In February 1920, however, he intervened in a domestic dispute between his sister and her husband, immediately after which several of his original symptoms recurred. He was readmitted into hospital, under Dr. S. H. Wilkinson, on April 21, 1920, with blepharospasm of the right eye, flaccid paralysis of the right arm, spastic paralysis of the right leg, and great difficulty in articulation. It was explained to him that, owing to the doubt in his mind as to the continued existence of brain disease because of the pain in the left side of his head, and knowing, as he did, that the left side controlled the right side of the body, his mind was just waiting for an excuse to produce the old symptoms again, and this excuse was afforded by the excitement of his intervention in the family squabble. Explanation was followed by persuasion and re-education, and in the course of an hour all the symptoms had disappeared.

In the following case continuous blinking was associated with deficient vision.

Case 8.—*Hysterical amblyopia and blinking following irritation by a lachrymatory shell.*—Private B., aged 44, was gassed in May 1916 by a lachrymatory shell. Next day he was able to carry on, but he constantly blinked and his vision became somewhat defective. The blinking and defective vision continued, but he did not go into hospital until April 1917.

The thorough examination of his eyes which was repeated in three different hospitals appears to have made him fear there was something serious the matter and to have led by suggestion to severer blindness, as the amblyopia and blepharospasm now became steadily worse. The case was diagnosed as disseminated sclerosis and subsequently as cerebellar tumour on account of the swaying gait, which was, however, simply an exaggerated result of defective vision. On admission to Netley in September 1917 his vision was 3/60 in both eyes; he was constantly blinking and had a staggering gait. He was too stupid for treatment by explanation to be effective, but suggestion with the aid of faradism applied to his eyelids caused the blinking to stop, and his vision and gait became normal for the first time for sixteen months. The next day the blinking had returned, but he again improved as a result of further suggestion, and a week later he was discharged to duty.

In the following case hysterical blindness occurred in a one-eyed man. The exciting cause would probably have been insufficient to have affected him had he not been living in perpetual fear that something might deprive him of the sight of his remaining eye.

Case 9.—*Partial hysterical blindness following shell-concussion in a one-eyed man.*—Sapper C., aged 28, lost his left eye in 1914 as the result of a shot-gun accident. When he enlisted in 1917 the vision of his right eye was 6/6. He went to France in May 1918 to work on the railway. In June 1918 an aeroplane dropped a bomb about twenty-five yards away, but did not hit him. The force of the explosion, however, was sufficient to dislodge his glass eye from its socket, and simultaneously his right eye became completely blind. The total blindness only lasted for a few days, but very defective vision persisted. He also noticed that erect objects, such as telegraph poles, appeared to be distorted. He passed through several hospitals, where the hysterical nature of the condition was recognised. He was still uncured when he was admitted to Seale Hayne Hospital, under Captain A. W. Gill, on October 26, 1918.

On examination there were no signs of disease in the eye. The pupil was of small size, and reacted briskly to light, but on attempting to focus an object he contracted the muscles of his neck, rotating his head to the right side, and alternating slight variations in the size of the pupil were evident. When he tried to read he held the book low down, and either to the left or right side. At a distance of two feet from the eye he could see objects distinctly, but anything nearer or beyond this limit was blurred and indefinite. The flinch reflex was completely absent. His mental attitude was one of great anxiety, and he was considerably depressed at the possibility of complete blindness.

He admitted that he went to France in considerable fear lest anything should happen to cause the loss of his one eye. The shock of the bomb explosion was sufficient to drive out his artificial eye, and this immediately suggested to him some damage to the other eye. Treatment directed towards relieving the patient's fears and encouraging him to relax his ciliary muscle was speedily followed by complete recovery, with return of the flinch reflex. Inequality of the pull of the ciliary muscle on the lens accounted for the distortion of upright images. On discharge from the hospital some weeks later his vision was 6/6.

In the next case the primary irritation of the eyes was caused by a sand-storm. It is of interest as showing two forms of hysterical blindness in one individual; in the right eye the motor element was alone affected, but in the left eye, which was more severely damaged in the sandstorm, so that the suggestion of blindness must have been stronger in connection with this eye, severe psychical blindness was present as well. The latter was almost as complete as the blindness of both eyes in case 1, and corresponding with this the pupil of the left eye was dilated compared with the other, and only responded very sluggishly to light.

Case 10.—*Hysterical blindness following sand-storm: twenty-six months' duration; cured by psychotherapy.*—Private F., aged 52, was caught in a sand-storm in December 1916, without any protection for his eyes. He developed

intense conjunctivitis and keratitis, with severe pain and photophobia, and was unable to open his eyes. Despite the fact that the inflammation and pain gradually disappeared, he was still unable to open his eyes when he was sent home from Egypt. In March 1917, no improvement having occurred, he was discharged from the Army as permanently unfit, with a disability of 100 per cent.

On February 10, 1919, he was admitted to Seale Hayne Hospital, under Captain A. W. Gill. He wore a pair of dark glasses, on removal of which he was found to have severe bilateral blepharospasm. With explanation and persuasion the spasm of the orbicularis muscles and the spasm of convergence, which was also present, were speedily overcome. It was then found that the left eye was completely blind, so that the patient could not distinguish light from darkness and could face the sun without blinking. The vision of the right eye was very defective, but he could pick out large objects and name some of them correctly. The pupils were unequal in size, the left being slightly the larger. Reaction to light was normal on the right side, but very sluggish on the left. Reaction to accommodation was irregular, but was sometimes brisk in both eyes. The blink reflex was present on the right side, but was completely absent on the left. Ophthalmoscopic examination showed that the retinæ were normal. There was no conjunctivitis, but slight keratitis was present in both eyes, being most marked in the left. Captain R. Jaques, who examined him for us, reported: "I can find nothing to account for the defect in vision. The corneal nebulæ are not sufficient for this."

Complete recovery followed psychotherapy: the pupils became equal in size and reacted normally, and a brisk flinch reflex was present on both sides. In the case of the right eye all that was required was to teach the patient to regulate the action of the ciliary muscle, relaxing it for distant objects and contracting it for near objects. In the case of the left eye it was necessary first of all to convince him that he would be cured, and then to encourage him to look with it.

There has been much discussion in the past as to whether homonymous hemianopia can ever be hysterical. Several cases, however, have been described. The following case is of interest, as hemianopia of one eye was associated with partial blindness of the whole field of the other eye.

Case 11.—*Hysterical blindness of one eye and hemianopia of the other cured by psychotherapy.*—Bombardier U., aged 25, developed severe conjunctivitis as the result of a bombardment with mustard-gas shells on March 21, 1918. After three weeks' treatment he was able to open his eyes, but found that he was almost completely blind in the left eye, being able to see only a white mist, while with the right eye he could see imperfectly. He was admitted to Seale Hayne Hospital, under Captain A. W. Gill, on November 5, 1918. The eye specialist's report accompanying him stated " left eye completely blind, right eye 6/24."

On admission there was no conjunctivitis and no sign of corneal scarring. The pupils were equal in size, and reacted sluggishly to light and accommodation. The left eye was completely blind, and the flinch reflex was absent. With the right eye he could see distant objects clearly. On attempting to read with the right eye he held the book slightly to the right side so that the light from the book fell on the nasal half of the right retina, the temporal half of which appeared to be blind. The flinch reflex was abolished, except when the direction of the blow was towards the nasal half of the right retina. Peripheral vision as tested with the moving finger was extremely defective in the right eye. Ophthalmoscopic examination showed the presence of a tiny foreign body embedded in the lens of the left eye, but no other organic changes were apparent. As the result of psychotherapy, complete recovery resulted in each eye, both for near and distant objects, and the flinch reflex was completely restored.

The presence of a small foreign body in the left lens, which had apparently been driven in some time before, had given rise to no visual trouble at the time, beyond making the patient aware that the vision of the left eye was less good than that of the right. This no doubt accounted

for the more complete hysterical loss of vision in this eye after the gassing. Believing that he was completely blind in the left eye, he appears to have subconsciously come to the conclusion that he would necessarily be blind to everything on his left side, and in this way the loss of the nasal half of the field of vision of his right eye was suggested.

The next three cases have no connection with the war, although two of them occurred in soldiers. For different reasons in each case, the patient had unconsciously taught himself to ignore the retinal impressions produced by one eye to such an extent that it had become blind, the condition being exactly analogous to the hysterical blindness already described. In the first case the patient taught himself to see once more with the blind eye when he began to lose the sight of the other as a result of optic atrophy. In the other two cases vision was restored by psychotherapy.

Case 12.—*Hysterical blindness of left eye; recovery of left eye following loss of sight of right eye caused by syphilitic optic atrophy.*—John P., aged 40, received a blow over the left eye in 1915; he could not open it, but on raising the lid he found he was blind. During the next eight months he regained the power of the levator palpebræ superioris, but he could still only see with his right eye. He was able to carry on with his occupation as a brass-worker until August 1919, when he found that vision with his right eye was becoming blurred by an orange-coloured mist. He now covered his right eye with a shade, and quickly taught himself to see almost perfectly with his left eye, which he had thought was blind. When he came under observation in October 1919 it was found that the recent loss of vision in his right eye was due to advanced optic atrophy, and the slight deficiency in the left eye was due to slight changes of the same nature. The right pupil reacted to accommodation but not to light; the left pupil responded to both. There were no other physical signs of disease. The Wassermann reaction of both blood and cerebro-spinal fluid was positive, and 54 lymphocytes per c.mm. were found in the latter, together with slight excess of globulin. The optic

atrophy was obviously of syphilitic origin, but there were no signs either of general paralysis or tabes. Anti-syphilitic treatment was given, but no change in vision resulted.

The blindness of the left eye following the blow was suggested by the pain and bruising, which prevented the patient from raising the lid. Four years later loss of sight in his other eye prompted him to try to see with his left eye, and to his surprise he found he could soon see fairly well. In order to abolish the blurred image produced by the right eye he covered this up. The slight deficiency in vision which persisted in the previously blind eye was not hysterical like the original total blindness, but was caused by a slight degree of optic atrophy.

In the next case the patient had a congenital defect of one eye, which led to deficient vision. He had learnt to neglect what he saw with this eye, but still retained some vision in it, when at the age of fifteen, for no obvious reason, he became almost totally blind in it. The blindness was suggested by the defective vision caused by the congenital abnormality, and was cured by psychotherapy. It could thus be correctly regarded as hysterical.

Case 13.—*Combined hysterical and organic blindness in the right eye ; hysterical element cured by psychotherapy after four years.*—Private M., aged 19, had never seen as clearly with his right eye as with the left. In 1914, while working as a clerk, he noticed that the right eye was becoming blind, and in February 1918, when he enlisted, he could only just distinguish light from dark with it. Captain R. Jaques found a persistent hyaloid artery passing from a posterior polar cataract to the optic disc. With very little persuasion he was taught to use the right eye again so well that he could read with it when the other was covered without difficulty. The pupil reactions were present in each eye, but the flinch reflex was totally abolished until vision was restored, when it became quite normal.

A severe squint dating from childhood in a man of 25 would not at first be regarded as likely to be hysterical,

(*a*) Before treatment. (*b*) After treatment by re-education.

FIG. 24.—Internal strabismus of nineteen years' duration.

But in the following case, not only the squint, but also the associated loss of vision in the affected eye, appear to have been of this nature. It is well known that when diplopia develops in adults they unconsciously train themselves to ignore one image; the same thing occurs in children who develop a permanent squint. The eye which ceases to be used in this way becomes more or less completely blind. After it has persisted for several years it is generally held that the blindness is permanent, and that even if an operation is performed to bring the eyeball into a central position, no improvement in vision is likely to occur. Moreover, in the event of an injury occurring to the sound eye sufficient to cause blindness, the previously squinting eye is generally said to be only capable of recovery if the squint has not been in existence for more than six or seven years.

Case 14.—*Hysterical blindness in a squinting eye; improved by psychotherapy after persisting for twenty years.*—Rifleman B., aged 25, at the age of six was frightened by a performing bear, and his mother states that he has squinted inwards with the left eye ever since, although she is confident that there was no squint prior to this event. On admission to Seale Hayne Hospital, under Captain A. W. Gill, on July 19, 1918, with hysterical paralysis of the right hand, which was cured the following day, the left squinting eye was found to be almost completely blind (fig. 24 (*a*)). There was no paralysis of any of the ocular muscles. Captain Jaques found + 3 diopters of hypermetropia in each eye.

There is no adequate explanation why squinting should result from an error of refraction which is equal in the two eyes. It seemed probable, therefore, that the squint was really a result of the fright caused by the bear. This might have caused an hysterical convulsive seizure, associated with squinting, as is so often the case in the convulsions of children. It was assumed that the squint had become perpetuated as an hysterical symptom, and an attempt was therefore made to treat both the squint and the blindness by psychotherapy. The patient was quickly taught to keep the two eyes nearly parallel (fig. 24 (*b*)). He was then

shown how to use his left eye, and at intervals the right eye was kept covered in order that the other should be in constant activity. We did not, however, succeed in curing the squint completely, although the patient could overcome it whenever he tried, and vision had so far recovered in the formerly blind eye that the patient could read large type when the other was covered.

The disturbances in vision so far described were independent symptoms, and filled the whole clinical picture. Hysterical blindness may also occur in one eye associated with paralysis of the same side, as in the following case, the graphic description of which we owe to Carré de Montgeron (*vide* frontispiece).[7]

Hysterical hemiplegia and hemianæsthesia with total blindness, immobility, anæsthesia, and loss of conjunctival and corneal reflexes of the corresponding eye.—On December 24, 1717, Marie-Jeanne, the 27-year-old wife of François Stapart, of Epernai, was seized with "apoplexy." After three days of fighting between life and death, the attack left her with paralysis and loss of sensation affecting the whole of the left side. "Of all the parts involved in this sad malady," wrote Montgeron, "the eye was the most seriously affected; the roots of the optic nerve of the left side were entirely enveloped in the obstruction of the brain; and as this nerve is the immediate organ of vision, the obstruction which deprived it of all action rendered it absolutely incapable of perceiving light. At the same time, the other nerves which serve for the movements and procure the sensibility both of the globe and the eyelids were equally completely obstructed; thus the eye and the lids lost all sensation and remained immobile. . . . The insensibility was so great that one could easily put one's finger between the lids and touch the eyeball without causing any movement or pain." That is to say, she was not only completely blind in the left eye, but the orbicularis was paralysed, the lids, conjunctiva, and cornea were anæsthetic, and the conjunctival and corneal reflexes were abolished. In the next six months the patient gradually regained the use of

her arm and leg, but the condition of the eye had remained unaltered, when nearly ten years later she had a second attack, which again affected her left side, and from which she again recovered. She was scarcely convalescent when she was struck down a third time, but on this occasion the paralysis of the left side was complete. The arm hung useless by her side, and she could only move with difficulty, dragging her useless left leg behind her. The muscles of the arm and leg became atrophied, and the skin œdematous. Being convinced that no human resources could help her, and recalling how she had heard that one Anne Angier had been cured of paralysis, which had affected her for twenty-one years, as a result of the intercession of M. Rousse, she determined to visit his tomb in the Chapelle de Ste. Anne at Avernai. "She was fortified in this resolution by a dream, which she took for a message from heaven. It seemed to her that she had been transported in the night to the tomb of M. Rousse, and that there the most perfect recovery had suddenly been vouchsafed to her."

On May 16, 1728, in spite of the opposition of the church authorities, because Rousse had not been canonised, she visited his tomb. Here she was suddenly seized with violent pain in her hand and eye; strength returned to her left hand so that she could join it with the right in prayer, and to her leg so that she could kneel. The eye which had been blind for ten and a half years, recovered its sight, its power of movement, and its sensibility. The recovery from the hemiplegia, hemianæsthesia, and blindness was complete, and there had been no relapse when Carré de Montgeron wrote his *Vérité des Miracles* nineteen years later.

In the following case it was easy to recognise that the blindness in one eye was the direct result of unconscious medical suggestion, whilst the patient was being examined on account of the hysterical hemiplegia for which he had been admitted into hospital. Being paralysed on his left side, he at once came to the conclusion that his vision was investigated because it was expected that the left eye

would be affected, and this was sufficient to suggest impaired vision.

Case 15.—*Hysterical blindness of left eye associated with hysterical hemiplegia cured by counter-suggestion.*—In October 1917, following a blow on the right side of the head, Pensioner S. developed left-sided facial paralysis and weakness of the left arm and leg. He was unconscious at first, and remained in bed until May 1918. He was discharged from the army with 100 per cent. disability in June 1918, and was admitted to Seale Hayne Hospital on March 4, 1919.

Complete left hemiplegia of the flaccid variety was present. All reflexes were normal, and the paralysis was typically hysterical in character. When the fields of vision were examined, it was found that the left eye was partially blind. The patient expressed great anxiety on this score, as he had not noticed it before. It was at once recognised that the loss of vision was caused by the suggestion of the examination, and by counter-suggestion the blindness was just as quickly removed. The hemiplegia was subsequently cured by psychotherapy.

Narrow and Spiral Fields of Vision in Hysteria, Malingering, and Neurasthenia.—Retraction of the field of vision has been regarded as the most characteristic "stigma" of hysteria since Charcot first drew attention to it in 1872. Janet [8] considered it to be "the emblem of hysterical sensibility in general," and it led him to describe hysteria as a condition due to "retraction of the field of consciousness." Among ophthalmologists de Schweinitz [9] regards this sign as "a permanent stigma" of hysteria, and believes its value as an aid to diagnosis to be "exceedingly great." But Babinski [10] found no change in the field of vision in any of a series of a hundred consecutive cases of hysteria, examined in great detail by methods which excluded the possibility of suggestion. Morax, [11] a former assistant of Charcot, from whom he learnt the supposed significance of the retracted field of vision, at first continued to find it in almost every patient suffering from hysterical symptoms. But over twenty years ago he became a convert to Babinski's

views, and since then he has not seen a single case of hysterical retraction of the field of vision. As it is almost impossible to avoid suggesting a narrow field of vision with the perimeter in highly suggestible individuals, he estimates the fields with the finger or other familiar object in hysterical patients, and although this may appear at first to be less accurate, it has the great advantage of making it easy to avoid suggesting abnormalities in the course of the examination.

In spite of the comparatively frequent occurrence of the

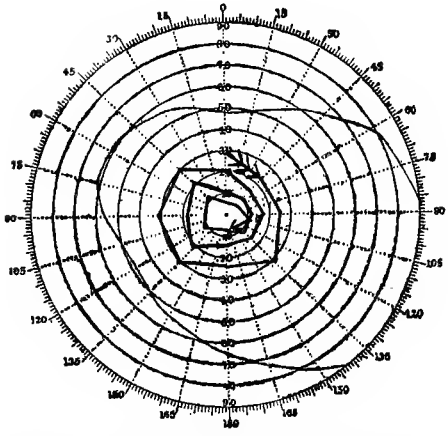

FIG. 25.—Without-inward spiral field of vision in right eye in case of hysterical paraplegia.

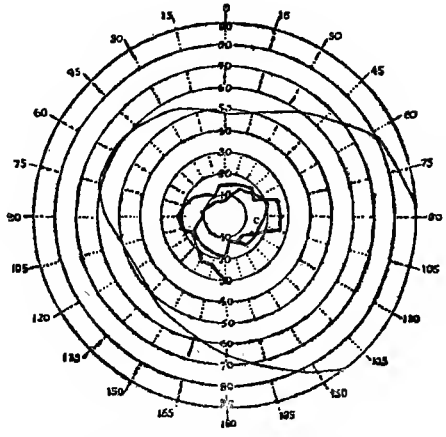

FIG. 26.—Spiral field of vison in hysteria. (After Purves Stewart.)

various hysterical disturbances of vision I have already described, I have never seen patients with hysterical symptoms affecting other parts of the body who spontaneously complained of disabilities resulting from a narrow field of vision, however closely they were cross-examined on the subject. But if a narrow field of vision is produced by testing with the perimeter, the patient may subsequently complain of considerable inconvenience as a result of the cutting off of his peripheral vision.

In association with Major J. L. M. Symns [18] I examined numerous soldiers suffering from various war neuroses, who were abnormally suggestible as a result of the stress

and strain of active service, some, but not all, of whom were suffering from gross hysterical symptoms. We never found any retraction of their field of vision until the perimeter was used. But the perimeter invariably resulted in the suggestion of a narrowed field, however carefully it was used. Moreover, if the examination was continued after the first field was marked out, a spiral field was always obtained (fig. 25) identical with that which has hitherto been regarded as a stigma of hysteria (cf. fig. 26). We believe that the reason why a spiral field of vision, which is the

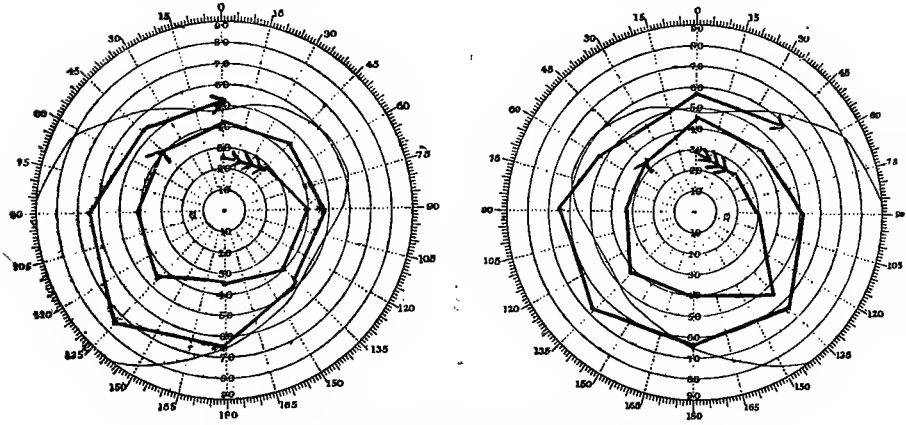

FIG. 27.—Spiral fields of vision in case of hysterical tremor.
(*a*) Left eye: within-outward spiral field.
(*b*) Right eye: within-outward spiral field.

natural result of continued suggestion, has not been found in a larger proportion of hysterical cases showing a narrow field of vision is simply because it has not been looked for, the examiner being content when he has marked the limit of vision a single time in each direction.

It has generally been taught that a spiral field of vision is a result of fatigue, and it has even been stated that it is more frequently a symptom of neurasthenia than of hysteria. We have found that this is not the case, the *inward* spiral, which has hitherto alone been described, being a result of the method employed in using the perimeter. An *outward* spiral is always obtained, instead of an inward one, if the

white disc of the perimeter is moved outwards instead of inwards, as is commonly done (fig. 27). In the description of the perimeter given in four books on ophthalmology, two on neurology, and one on clinical methods, which we consulted, instructions are given to move the disc from without inwards. By varying the direction in which the disc was moved we could produce an inward spiral one day and an outward spiral another day with the same eye, or an inward spiral with one eye and an outward spiral with the other at the same time (fig. 28). There is no question of

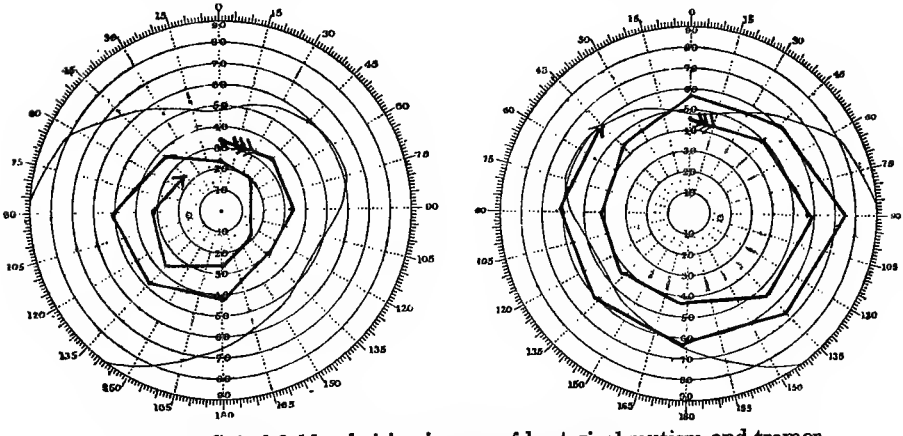

FIG. 28.—Spiral fields of vision in case of hysterical mutism and tremor.
(*a*) Left eye: without-inward spiral field.
(*b*) Right eye: within-outward spiral field.

any special suggestions made by us in our method of using the perimeter, as identical results were obtained by other observers, who marked out the fields of vision for us without knowing the nature of the cases or the object we had in view in obtaining the tracings.

In the observations on "experimental malingering," which I described in chapter I, we asked twenty-seven individuals, who were pretending to be paralysed on the right side, and none of whom had mentioned any disturbance in vision when detailing their symptoms, whether they could see as well with the right eye as the left: seven replied that they had noticed some impairment of vision in the

right eye. On testing the field of vision with a finger no narrowing was observed, and they explained that the deficiency they had spoken about was a blurring or general loss of clearness of vision. But when tested with a perimeter all of the seven showed a narrow field on the right side, and one had a slighter narrowing on the left side. In the only two cases in which it occurred to us to continue the investigation after the first field had been marked out a spiral was obtained (fig. 29), which was identical in character with that supposed to be characteristic of hysteria.

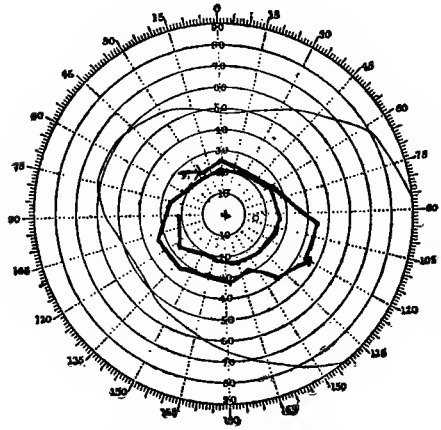

FIG. 29.—Spiral field of vision in case of "experimental malingering."

Ocular Aura in Hysterical Fits.—I believe that hysterical fits are much more common than is generally supposed, and that many such cases are diagnosed as epilepsy and treated for years with bromides. It has been said that a definite aura is uncommon in hysteria but common before true epileptic fits. I am convinced that this is incorrect, and that some kind of warning invariably precedes an hysterical attack. It is, indeed, this "warning," which acts as the suggestion, which is the exciting cause of each fit. A symptom which has once produced an attack will be likely to produce another whenever it recurs, because it subconsciously reminds the patient of his earlier attack, and thus suggests that another will occur.

I have seen several cases of hysterical attacks which were preceded by a visual aura. Most of the cases in soldiers dated from a period of strenuous activity in the glare of Mesopotamia. In the following case, in which the attacks

consisted of headache and loss of consciousness, and not of epileptiform convulsions, an understanding of the psychological processes involved resulted in immediate recovery from a very incapacitating condition which had persisted for two years.

Hysterical photophobia with attacks of headaches and loss of consciousness.—Captain S. was wounded in the head in 1915. He was temporarily paralysed on the right side, and suffered from violent headaches, which culminated in loss of consciousness. He gradually improved, and no more attacks occurred after the end of 1916. He went back to France in 1917. Soon afterwards he had some acute mental disorder, for which he was in hospital for five months; after this he was invalided out of the service, but was fit enough to be sent to Greece on diplomatic duty. In 1918 he went to Salonica for an operation to be performed on his heel. Some ether appears to have been accidentally dropped into his eyes during the operation, as when he came round from the anæsthetic he found his eyes were so painful that he could not open them, and he thought he had lost his sight. At the end of a month he could see a little, but he could not read, and had always to wear the darkest glasses he could get, as directly his eyes were exposed to the daylight he had a violent headache, which was followed in a few minutes by complete loss of consciousness.

I first saw him in February 1920. It was very difficult to examine his eyes, as he kept them tightly closed when the dark glasses were removed. Even when he was wearing them he never opened his eyes widely, but he could see enough to get about without assistance. He was very depressed about his condition. His arm and leg were quite well, and there were no physical signs of organic disease. There was a slight depression of the skull in the region of his wound, but it was not tender, and there was no bony deficiency.

I explained to the patient that when the removal of the bandages from his eyes for the first time after they had been injured was followed by a headache, he subconsciously associated this with the headaches which had followed the

wound to his head, and that he had consequently suggested to himself the same sequel—loss of consciousness. The attacks of headache followed by unconsciousness now became associated in his mind with his eyes instead of with the original injury to his head. Consequently an attack occurred every time he removed his dark glasses, and for this reason he feared to face the light. The blepharospasm and deficient sight were produced in the way I have already described in connection with the hysterical disorders of vision caused by gassing. Having discussed this explanation with him in great detail, I promised him he would recover completely when he came to me for treatment the next day. He arrived fully convinced he would be cured, having thoroughly grasped the explanation I had given him of the origin of his symptoms. Without difficulty I persuaded him to remove his dark glasses, keep his eyes widely open, and accommodate his vision for near and distant objects. For the first time for two years he was able to keep his eyes open without getting a headache, and an hour later he went for a walk in the sun without glasses. In the five months which have since elapsed he has had no further attacks of headache and loss of consciousness, and he is shortly returning to his diplomatic duties.

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